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DISSERTATION

ON

AN OVERVIEW OF GUILLAIN-BARRÉ SYNDROME WITH REFERENCE TO CLINICAL FEATURES AND PROGNOSTIC OUTCOME

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CONTENTS

INTRODUCTION

Guillain-Barré Syndrome is the commonest cause of acquired demyelinating disorders affecting the peripheral nervous system in any part of the world. It is a spectrum of illness of diverse etiology with a common pathological process. It is a non-seasonal illness affecting persons of all age groups.

The severity of Guillain-Barré Syndrome varies from mild weakness to total paralysis and respiratory failure, sometimes leading to death.

Proper understanding of pathology, clinical presentation, appropriate investigations and interventions when needed may save these patients from mortality and severe morbidity.

Thanjavur Medical College and Hospital predominantly covers rural population of Tamilnadu. It offers medical management to all economic status of people, all religions and all age groups. It is more ideal to conduct a study in this institution.

The diagnosis of Guillain-Barré Syndrome is made predominantly by clinical methods and aided by investigations like cerebrospinal fluid analysis and electrodiagnostic studies.

AIMS OF THE STUDY

1. To evaluate the types and antecedent events of Guillain-Barré Syndrome

2. To find out the incidence of Guillain-Barré Syndrome

3. To analyze the temporal profile of illness and its clinical features

4. To assess the severity of illness with reference to cerebrospinal fluid analysis and electrodiagnostic studies

5. To assess the prognostic outcome with electrodiagnostic studies

HISTORICAL REVIEW

History of Guillain-Barré Syndrome divides into

1. **1843 – 1916: DESCRIPTION OF THE SYNDROME [32]**

1843 – Robert Graves recognized that peripheral nerve disease could lead to paralysis.

1859 – Jean Baptiste Octave Landry found the paralytic disease without central lesions might be due to peripheral nerve involvement[1, 28].

1864 - Dumenil in Rouen reported first pathologic demonstration of nerve disease.

1880 – First autopsy description of lymphocytic inflammation by Leyden.

Early 1890s – Osler used the term acute febrile polyneuritis[1].

2. **1916 -1969: SETTING BOUNDARIES[32]**

1916 - Georges Guillain, Jean-Alexandre Barré and Andre Strohl described two paralyzed French soldiers[1,48]. Their novel contribution was the demonstration of elevated spinal fluid protein without cells[28].

1960s - Advent of Electrodiagnosis – Landmark in diagnosis of Guillain-Barré Syndrome. Hay maker and Kernohan-definite identification of demyelination[1].

3. 1969 – PRESENT MECHANISMS AND TREATMENTS[32]

1969 - Asbury, Amason, Adams demonstrated analogy between Guillain-Barré Syndrome and Experimental Allergic Neuritis (EAN) in animals[1].

Mid 1980s – First effective immunotherapy – the new technique of plasmapheresis. Second – infusion of intravenous immunoglobulin.

Yuki – First demonstrated molecular mimicry in post-Campylobacter Guillain-Barré Syndrome.

REVIEW OF LITERATURE

DEFINITION

Guillain-Barré Syndrome can be defined as an acquired, immunologically mediated or autoimmune, acute, inflammatory, demyelinating, polyradiculoneuropathy (AIDP) [28]. The most frequent cause of acute flaccid paralysis worldwide, [4, 30] are also among the most dramatic neurologic emergencies.

SYNONYMS[5]

Acute post - infective polyradiculoneuropathy

Acute infectious polyneuritis

Landry - Guillain-Barré - Strohl syndrome

Post - infective polyneuritis

INCIDENCE

- Reported incidence rate has varied from 0.4 to 1.7 cases per 100,000 persons per year (Ropper et al 1991)[1,12,42,44]
- Non – seasonal, non-epidemic illness, affects persons of all ages[1,25]
- Reports from India and China indicate peak incidence between July and October[28]
- Females are slightly more susceptible[1]

CLASSIFICATION OF THE GUILLAIN-BARRÉ SYNDROME SUBTYPES[1, 35, 44, 48]

1. Acute inflammatory demyelinating polyradiculoneuropathy
2. Acute motor axonal neuropathy
3. Acute motor sensory axonal neuropathy
4. Miller – Fisher Syndrome
5. Acute pandysautonomia
6. Sensory Guillain-Barré Syndrome
7. Cervico-brachial-pharyngeal, often with ptosis
8. Bilateral facial or abducens weakness with distal paresthesias

DIAGNOSTIC CRITERIA OF AIDP (ASBURY AND CORNBLATH 1990)[4, 12,23,44,48]

FEATURES REQUIRED FOR DIAGNOSIS

- Progressive weakness in both arms and both legs
- Areflexia

FEATURES STRONGLY SUPPORTING THE DIAGNOSIS

- Progression of symptoms over four days to four weeks
- Relative symmetry of symptoms
- Mild sensory symptoms or signs
- Cranial nerve involvement especially bilateral weakness of facial muscles
- Recovery beginning 2 - 4 wks after progression ceases

- Autonomic dysfunction
- Absence of fever at the onset
- Elevated concentration of proteins in the cerebrospinal fluid with less than 10 mononuclear cells per cubic mm
- Typical electro diagnostic features

FEATURES MAKING THE DIAGNOSIS DOUBTFUL

- Sensory level
- Marked persistent asymmetry of symptoms and signs
- Severe or persistent bladder or bowel dysfunction
- More than 50 mononuclear cells per cubic mm in cerebrospinal fluid
- Presence of polymorphonuclear leucocytes in cerebrospinal fluid

FEATURES EXCLUDING THE DIAGNOSIS

- Diagnosis of Botulism, Myasthenia, Poliomyelitis or Toxic neuropathies
- Abnormal porphyrin metabolism
- Recent diphtheria
- Purely sensory syndrome without weakness
- Features clinically consistent with lead neuropathy
- History of hexacarbon (volatile solvent) abuse[46]

CLINICAL MANIFESTATIONS

Guillain-Barré Syndrome is typically a monophasic illness[50]. Approximately, two thirds of patients report a preceding event, 1- 4 weeks before the onset of neurological symptoms (Govoni and Granieri 2001)[8].

ANTECEDENT EVENTS[1, 5, 12,19,28,48]

1. Respiratory illness 58%
2. Gastrointestinal illness 22%
3. Respiratory and gastrointestinal illness - 10%
4. Surgery – 5% (Winer et al)
5. Vaccination - 3%
    - 1976 A / New Jersey influenza vaccine (Schonberger et al)
    - Tetanus toxoid
    - Diphtheria toxoid
    - Rabies
    - Oral Polio vaccine
    - Small pox vaccine
    - Meningococcal vaccine

- Anthrax vaccine

6. Serological evidence of specific infectious agents

- Campylobacter jejuni - 26%
- Cytomegalovirus - 15% (Visser et al)
- Human immuno deficiency virus - 1
- Ebstein - Barr virus - 8%
- Mycoplasma pneumoniae – 10%
- Varicella-Zoster virus
- Hepatitis A and B
- Hemophilus influenza
- Childhood Exanthems (Cornblath et al)

7. Others

- Drugs – Streptokinase, Suramin, Gangliosides, Heroin
- Hymenoptera stings
- Hodgkin's lymphoma
- After solid organ or bone marrow transplantation
- Systemic Lupus Erythematosus

ROSS and BURY'S DIVISION[32]

1. Stage of invasion
2. Progression and plateau phase
3. Stage of regression

Paresthesias and slight numbness in the toes and fingers are the earliest symptoms[1, 3, 4, 5]. The fairly symmetrical weakness of the lower limbs ascends proximally over hours to several days to involve arm, facial and oropharyhgeal muscles, and in severe cases respiratory muscles - Landry's ascending paralysis[1, 14, 16]. Trunk, intercostal, neck and cranial muscles are affected later. Hyporeflexia or areflexia are invariable features[5, 11, 48].

Cranial nerve involvement ranges from 45% - 75%[48]. Facial paresis[1, 9, 22, 48,] usually bilateral is found in atleast one half of patients. If the weakness of the face is out of proportion to that of the limbs, Bannwarth syndrome or Lyme disease should be considered[5]. Bulbar palsy and weakness of the muscles of mastication are the next most common cranial nerve abnormalities[31]. Involvement of extraocular muscles occurs in 10 percent of patients[5].

Sensory loss is limited frequently to distal impairment of vibration sense and joint- position sense (Winer et al 1988)[14]. Interscapular or low back pain with radiation into the legs is most common and may require short term opiate analgesia[5,15]. Respiratory failure of sufficient severity to require assisted ventilation occurs in 30

percent of patients, although milder degrees of respiratory muscle involvement are much more common.

Autonomic dysfunction had been reported in 65% of Patients (Zochodne 1994)[1, 5, 35,46,48,50]

Signs of decreased sympathetic activity include orthostatic hypotension, anhidrosis. Signs of increased sympathetic activity include episodic or sustained hypertension, sinus tachycardia, tachyarrhythmias, episodic diaphoresis, and acral vasoconstriction.

Signs of decreased parasympathetic activity include urinary retention, gastrointestinal atony, iridoplegia. Signs of increased parasympathetic activity include sudden episodes of bradycardia, heart block, asystole.

ECG changes include T - Wave abnormalities, ST – Segment depression, QRS widening, QT prolongation, various forms of heart block.

Forms of Guillain-Barré Syndrome precipitated by both Campylobacter and Cytomegalovirus show delayed recovery compared to cases unassociated with these two infections[5]. Optic neuritis and pyramidal tract signs are rare manifestations[5].

AXONAL FORMS

1. Acute Motor Axonal Neuropathy (AMAN)

2. Acute Motor Sensory Axonal Neuropathy (AMSAN)

Axonal forms of Guillain-Barré Syndrome cannot be differentiated form AIDP without electrophysiology or extensive pathologic data[11, 41]. Most patients with AMAN recover as rapidly as patients with AIDP. Antecedent C. Jejuni infection was found in 76% of AMAN patients[3, 5, 10]. AMSAN –All patients had a hyperacute course, profound quadriparesis and required prolonged ventilator support. Prognosis is unusually poor[32, 50].

DIFFERENTIAL DIAGNOSTIC CONSIDERATIONS[1, 8, 12, 48]

1. **ACUTE NEUROPATHIES**

Hepatic porphyrias

Critical illness neuropathy

Diphtheria

Toxins

Arsenic, thallium, organophosphates, lead neurotoxic fish and shellfish poisoning (ciguatoxin, tetrodotoxin, saxitoxin)

Buckthorn

Tick paralysis

Vasculitis

Inflammatory meningoradiculopathies

Lyme disease

Cytomegalovirus lumbosacral radiculomyelopathy

Acute axonal alcoholic polyneuopathy

2. **DISORDERS OF NEUROMUSCULAR FUNCTION**

Botulism

Myasthenia gravis

3. **MYOPATHIES**

Hypokalemia

Hyopophosphatemia

Rhabdomyolysis

Intensive care Myopathy

4. **CENTRAL NERVOUS SYSTEM DISORDERS**

Poliomyelitis

West Nile virus poliomyelitis

Rabies

Transverse Myelitis

Basilar artery thrombosis

LABORATORY FINDINGS

CEREBROSPINAL FLUID ANALYSIS

Cerebrospinal fluid is under normal pressure and is acellular or contains a few lymphocytes, 10 - 50 cells per cubic mm, predominantly lymphocytes[1, 39]. (albumino – cytological dissociation). In the first week of neurological symptoms, cerebrospinal fluid protein may be normal[8]; begins to rise with a peak in 4 to 6 weeks (100 to 1000 mg/dl) [48]. Persistent pleocytosis suggests an alternative or additional diagnosis such as neoplastic meningitis, HIV or Lyme Infection (Cornblath et al 1987) [1].

In 10% of cases, cerebrospinal fluid protein remains normal throughout the illness. The increase in cerebrospinal fluid protein is probably a reflection of the wide spread inflammatory disease of the nerve roots[1, 12, 23]. Impaired cerebrospinal fluid circulation due to increased protein may lead to papilloedeama and pseudotumor cerbri (Weiss et al 1991)[1, 28].

ELECTRODIAGNOSTIC STUDIES

Abnormalities of electrophysiological studies are found in approximately 90% of established cases[1, 5,6,48].

The most common abnormalities include[24]

1. Prolonged distal motor and F-Wave Latencies.
2. Absent or impersistent F waves.
3. Conduction block.

4. Reduction in distal CMAP amplitudes with or without temporal dispersion.

5. Slowing of motor conduction velocities.

Needle EMG[48] initially shows decreased motor unit recruitment; if axonal degeneration occurs 2 - 4 weeks after onset, fibrillation potentials appear. Electrodiagnostic parameters are the most reliable indicators of prognosis. A distal CMAP amplitude of less than 20% of the lower limits of normal was associated with poor outcome in the North American Guillain-Barré Syndrome Study.

Electro diagnostic studies in acute motor axonal neuropathy include reduced CMAP amplitudes, normal motor distal latencies and normal conduction velocities[5, 24, 48].

Electro diagnostic studies in acute motor sensory axonal neuropathy include markedly reduced or absent CMAPS with distal supramaximal stimulation without conduction delay; absent SNAPS, abundant fibrillation potentials, persistently inexcitable motor nerves on needle EMG.

SEROLOGICAL TESTS[12, 48]

Type	Antibody Target	Usual Isotype
AIDP	**GM_1 Most common**	**IgG (polyclonal)**
Axonal Variants	**GD_{1a}, GM_1, GM_{1b}, GalNAc – GD_{1a} (<50% for any)**	**IgG (polyclonal)**
MFS	**$GQ1_b$ (>90%)**[8]	**IgG (polyclonal)**

Complement fixing antibodies to peripheral nerve myelin P_2 are present in acute phase of Guillain-Barré Syndrome. Anti galactocerebroside antibodies are present in patients with precedent Mycloplasma infection[48].

OTHERS

Lumbosacral spinal MRI may demonstrate gadolinium enhancement of the cauda equina roots. Abnormalities of liver function occur in <10% of patients, probably reflecting a recent or ongoing viral hepatitis[48]. Sedimentation rate is usually normal. Hyponatremia occurs[1, 48], especially in ventilated patients due to SIADH or excess of atrial natriuretic factor. Transient diabetes insipidus is rare[1]. Deposition of immune complexes may rarely lead to glomerulonephritis[1].

SPECIAL STAINS

Special stains like Luxol Fast Blue, Immunoperoxidase will show loss due to demyelination. Ultrastructural study through electron microscopy of peripheral nerve in Guillain-Barré Syndrome shows presence of mononuclear cells in axons.

PATHOLOGY

Virtually all cases of classic Guillain-Barré Syndrome show perivascular (mainly perivenons) lymphocytic infiltrates together with multifocal demyelination[1, 4, 13]. Intense inflammation may lead to axonal degeneration as a consequence of a toxic bystander effect[1, 48]. The inflammatory infiltrates consist mainly of class II – positive monocytes and macrophages and T lymphocytes[13]. In acute motor sensory axonal neuropathy extensive primary wallerian – like degeneration of motor and sensory roots and nerves without significant inflammation or demyelination is found (Lu et al, Feasby et al)[1,48].

PATHOGENESIS

A preceding infection may trigger an autoimmune response through molecular mimicry in which the host generates an immune response against an infectious organism that shares epitopes with the host's peripheral nerves[1, 5, 12]. Waksman and Adams demonstrated that experimentally induced peripheral nerve disease (Experimental Allergic Neuritis or EAN) is clinically and pathologically indistinguishable from Guillain-Barré Syndrome[1, 12, 48]. At the onset of disease,

activated T cells play a major role in opening the blood – nerve barrier[39]. T – Cell activation markers (1L-6, 1L-2, soluble 1L-2 receptor, 1FN-γ), TNF – α are increased in serum[4] (Hartung et al). Brostoff and colleagues suggested that the antigen in this reaction is a basic protein, designated P_2 found only in peripheral nerve myelin[1, 12].

Humoral factors participate in the autoimmune attack on peripheral nerve myelin, axons and nerve terminals as evidenced by[13, 48]

1. Immunoglobulins and complement can be demonstrated on myelinated fibers.
2. Miller – Fisher Syndrome and acute motor axonal neuropathy are strongly associated with specific antiganglioside antibodies.
3. Complement C_1 – fixing antiperipheral nerve myelin antibody can be detected in acute phase.
4. Plasmapheresis or immunoglobin infusion results in clinical improvement.

Earliest changes seen within days of onset consisted of deposition of complement activation products and membrane attack complex on the outermost Schwann cell surface followed by vesicular or myelin changes (Hasfer-Macko et al) [1,5]. Finally macrophages are shown to invade the periaxonal space leading to wallerian like degeneration of motor fibres.

The most attractive candidate targets are GM_1 and asialo-GM_1 like gangliosides, which are present in nodal and internodal membranes of motor fibres[34].

Acute motor sensory axonal neuropathy may be caused by a more severe immune injury triggered by axonal epitopes[48].

COMMON COMPLICATIONS OF GUILLAIN-BARRÉ SYNDROME[1, 5, 44, 48]

- Respiratory failure with or without bulbar paresis
- Dysautonomia
- Deep vein thrombosis
- Pulmonary embolism
- Chest infection
- Urinary tract infection
- Ileus
- SIADH
- Anxiety neurosis and depression

NEUROLOGICAL DISABILITY GRADING IN GUILLAIN-BARRÉ SYNDROME (Hughes et al 1978)[28, 44]

Grade 0 – Healthy

Grade 1 – Minor signs or symptoms of neuropathy but capable of manual work

Grade 2 – Able to walk without support but incapable of manual work

Grade 3 – Able to walk with a stick, appliance or support

Grade 4 – Confined to bed or chair-bound

Grade 5 – Requiring assisted – ventilation

Grade 6 – Dead

MODIFIED DISABILTY GRADING SCALE FOR GUILLAIN-BARRÉ SYNDROME (Modified form Hughes et al 1978)[28,44]

Grade 0 – Healthy

Grade 1 – Minor signs or symptoms

Grade 2 – Able to walk 5 meters without support

Grade 3 – Able to walk 5 meters with a stick, appliance or support

Grade 4 – Confined to bed or chair-bound (unable to walk 5 meters with support)

Grade 5 – Requiring assisted – ventilation (for atleast part of the day)

Grade 6 – Dead

TREATMENT

GENERAL MEDICAL ASPECTS

Supportive care and prevention of complications in intensive care units, of which respiratory failure and autonomic dysfunction are the most important, provide the best chance for a favorable outcome. During the progressive phase, respiratory and bulbar function, the ability to handle secretions, heart rate and blood pressure should be closely monitored[1, 20].

Respiratory failure requiring mechanical ventilation develops in upto 30% of patients with Guillain-Barré Syndrome. The mortality has fallen from 33% before introduction of positive pressure ventilation to the current rate of approximately 5-10% [48].

Signs of impending respiratory failure include[36, 48]

1. Deterioration in Forced Vital Capacity (FVC)
2. Declining maximal respiratory pressures
3. Tachyponea and decrease in arterial oxygen tension (PO_2 < 85mm Hg) reflecting pulmonary atelectasis

The strength of the neck muscles and trapezii, which share the same segmental innervations as the diaphragm, tends to parallel diaphragmatic strength. Ability to

reach 20 by single breath count generally corresponds to a vital capacity of greater than 1.5 L.

Predictors of future need of mechanical ventilation[1, 5, 48]

1. Rapid disease progression (onset to admission in less than 7 days)
2. Bulbar dysfunction
3. Bilateral facial paresis
4. Autonomic instability

Predictors of future respiratory failure 20 - 30 – 40 rule[48]

1. Vital capacity of less than 20 ml/kg or a decline by 30% from baseline
2. Maximal inspiratory pressure less than 30cm of H_2O
3. Maximal expiratory respiratory pressure less than 40cm of H_2O

Elective intubation should be performed when[17, 48, 49]

1. FVC falls below 12-15 ml/kg
2. FVC falls below 18 ml/kg with severe oropharyngeal weakness
3. Arterial PO_2 values <70mmHg with inspired room air

Indications of tracheostomy[1, 5]

1. When the need for mechanical ventilation > 2 weeks

2. Failure to clear the tracheobronchial air ways effectively

With tracheostomy and intensive care, the mortality from disease can be reduced to about 3% (Ropper and Kehne) [48]. During intubation, succinyl choline is not preferable due to upregulation of acetylcholine receptors on denervated muscles may causes lethal hyperkalemia[2]. Nondepolarizing agents may be problematic because of resistance and increased sensitivity. Etomidate is preferred, if oral intubation is done, because of the inherent hemodynamic stability.

OTHER MEASURES

Hypotension from dysautonomia is treated by intravenous volume infusion and by the use of vasopressor agents for brief periods[1]. Extremes of hypertension are managed by short-acting and titratable antihypertensive medications such as intravenons labetolol[1].

Prevention of electrolyte imbalance, gastrointestinal hemorrhage, noscomial infections are important in intensive care. Deep venous thrombosis and pulmonary embolism can be prevented by the use of subcutaneous heparin, low molecular weight heparin and calf compression devices[1, 12, 30].

Exposure keratitis is avoided by using artificial tears. Pressure – induced ulnar or fibular nerve palsies are prevented by proper positioning and padding. Physical therapy[1,18] is started early because it helps prevent contractures, joint immobilization and venous stasis. Psychological support and constant reassurance are essential[45].

SPECIFIC THERAPEUTIC INTERVENTIONS

Immunomodulation has been used to enhance recovery and avoid complications of Guillain-Barré Syndrome[28]

Treatment modalities include

1. Plasma exchange
2. Intravenous immunoglobulin therapy

SELECTION CRITERIA[28]

1. Time lapse between disease onset and assessment of the patient.
2. Disease severity.
3. Rate of disease progression.

Should be done as early as possible preferably during the first week. Patients with mild symptoms, slow or lack of disease progression do not require immunomodulation.

INDICATIONS

Patients who

1. Are unable to walk unassisted
2. Have bulbar paralysis
3. Are on the verge of respiratory failure or require ventilator assistance
4. Shows rapid disease progression within two weeks of disease onset

PLASMA EXCHANGE – ADVANTAGES[1, 5, 12,28,44,48]

1. Rapid onset of action
2. Usually safe and well tolerated
3. Few side effects
4. Effective in two-third of cases
5. Hastens recovery and the time taken to improve one clinical grade (Guillain-Barré Syndrome Study Group 1985)
6. Shortens the time on ventilator and decreases the chances of becoming ventilator dependent
7. Improves the poor prognostic factors
8. Decreases the percentage of cases with residual disability at 6 months

Predictors of responsiveness to plasma exchange treatment are the patient's age (young) and the preservation of CMAP amplitudes prior to instituting treatment[1, 47]. The advised regimen of plasma exchange removes a total of 200 – 250ml/kg of plasma in four to six treatments on alternate days. The replacement fluid is saline combined with 5% albumin.

COMPLICATIONS AND SIDE EFFECTS[1, 28, 44]

1. **Hemodynamic and cardiovascular**
   - Hypotension
   - Acute respiratory distress syndrome
   - Acute Cardiac Insufficiency
   - Myocardial infarction
   - Arrhythmias
2. **Complications due to vascular access**
   - Septicemia
   - Thrombosis
3. **Associated with replacement fluids / anticoagulants**
   - Allergic reactions
   - Hepatitis, HIV (FFP)
   - Citrate toxicity

- Bleeding

4. Depletion of Plasma Components

- Loss of clotting factors, globulins
- Loss of protein – bound drugs

5. Technical Complications

- Clotting in the tubing
- Air embolism
- Haemolysis
- Death (0-0.3%)

INTRAVENOUS IMMUNOGLOBULIN THERAPY [1, 12, 35,44,46,48]

IVIG contains normal polyvalent immunoglobulin G, derived from a large number of blood donors.

Possible mode of action of IVIG includes

1. Anti-idiotypic antibody effect
2. Blockade of Fc receptors on macrophages (interferes with antibody dependent cellular cytotoxicity)
3. Down regulation of immunoglobulin production
4. Down regulation of B Cells (CD_5 +B Cells by anti CD_5 antibodies)

5. Inhibition or attenuation of cytokine actions by anticytokine antibodies
6. Interference with T cell activation
7. Induction of T suppressor cells
8. Inhibition or neutralization of complement mediated effects
9. Virus neutralization

Safe in patients as young as 2 years to as old as 93 years of age[43]. Should be avoided in pregnancy. Miller Fisher Syndrome and Acute Pandysautonomia also respond to IVIG[28].

DOSE AND MODE OF ADMINISTRATION[1, 5, 12, 28]

- Adults – 400 mg/kg/day for 5 consecutive days, children- 1gm/kg/day for 2 days.
- Half - life of IVIG is around 23 days.
- Its beneficial effect may last for 2-9 weeks.

COMPLICATIONS AND SIDE EFFECTS[1,12,28,44]

- Headache including precipitation of migraine
- Back pain
- Fever
- Tachycardia

- Aseptic meningitis
- Rash (Erythema multiforme, purpuric erythema, maculopapular, petechial)
- Alopecia
- Eczema
- Reversible neutropenia
- Hemolysis
- Immune complex arthritis
- Thromboembolism
- Transmission of hepatitis C Virus
- Deep venous thrombosis
- Congestive cardiac failure
- Acute hypotension
- Oligaemic renal failure
- Cerebral vasospasm
- Anaphylaxis – in IgA deficient individuals

ADVANTAGES OF IVIG[1, 40, 44]

- Wide availability
- Does not require special equipment
- Can be given at any center
- Easily administered

- Can be used with ease in small children and aged patients with limited access or other contraindications for plasma exchange
- Few side effects
- No risk of HIV
- Rapid response
- Equally effective or slightly superior to PE
- 75% cases show good response to treatment

PLASMA EXCHANGE VS IVIG THERAPY[28]

Both PE and IVIG have emerged as equally effective treatment options in Guillain-Barré Syndrome. Around 66 – 75% patients respond.

IVIG is often preferred as the first line agent in children, elderly, Guillain-Barré Syndrome variants like MFS, Acute pandysautonomia, Guillain-Barré Syndrome with prominent sensory ataxia, pure motor forms.

COMBINED USE OF PE AND IVIG[28, 48]

PE and IVIG have precisely opposite effects. The body pool of immunoglobulins is depleted by PE and expanded by IVIG. Combined PE and IVIG may have a synergistic effect.

IVIG administration immediately after PE may prevent a rebound antibody synthesis to peripheral nerve myelin following removal of immunoglobulins and antibodies by PE[28].

2 recent studies using combined PE and IVIG therapy have observed only a slight increase in efficacy with the combined approach as compared to the use of IVIG or PE alone. Above two treatments can be repeated if a patient is clearly declining particularly if there is evidence of mainly demyelinating disease on the EMG and if the illness is not much longer than 3 weeks duration.

ROLE OF CORTICOSTEROIDS

Two randomized controlled studies, one with conventional - dose prednisolone and the other with high dose methylprednisolone have failed to demonstrate any beneficial effect[15, 28, 47] (Guillain-Barré Syndrome steroid trial group 1993). Steroids might increase the subsequent relapse rate (Hughes et al 1978).

ROLE OF GABAPENTIN[28]

Gabapentin, an antiepileptic drug, has been used effectively for different types of pain management. This study demonstrates that gabapentin has minimal side effects and is an alternative to opioids and NSAIDS for management of the bimodal nature of pain of Guillain-Barré Syndrome patients.

COURSE AND PROGNOSIS

By definition, patients should reach their maximum deficit within 4 weeks of onset, if the disease progresses for longer, it is classified as subacute or chronic inflammatory demyelinating polyradiculoneuropathy (SIDP, CIDP)[27,29].

Upto 30% of patients develop respiratory insufficiency and between 2-5% die of complications[10].

70% of patients complete their recovery in 12 months[8, 12], 82% in 24 months.

20% have still residual motor weakness 1 year later[1].

About 3% of patients may have a recurrence following recovery.

As a rule, older adults recover more slowly than younger ones and children and have more residual weakness[1, 37].

Poor Prognostic Factors (North American Guillain-Barré Syndrome study group) [5, 42, 48]

1) Older age > 60 years
2) Preceding diarrhoeal illness
3) Respiratory failure requiring ventilatory support
4) Rapid progression reaching maximum deficit in less than 7 days
5) Low amplitudes of distal CMAPS (20% of lower limit of normal or less)

6) Extensive spontaneous fibrillation in distal muscles indicating denervation

The mortality is 4 – 5% even for patients treated in specialist neurological units with PE or IVIG (PE / Sandoglobulin GBS trial group 1997)[33].

CAUSES OF DEATH[1,5]

EARLY STAGES

- Cardiac arrest due to dysautonomia
- Adult respiratory distress syndrome
- Pneumothorax
- Hemothorax
- Accidental machine failure

LATE STAGES

- Pulmonary embolism
- Medical complications of prolonged immobilisation
- Respiratory failure

RELAPSING FORMS

Recurrent Guillain-Barré Syndrome occurs in up to 3%, after an interval of many years (Loffel et al 1977)[1,5]. It may be due to a cyclic dysimmune state of the host or unidentified triggering factors acting on a vulnerable host.

The episodes may be precipitated by new infection, such as recurrent cytomegalovirus exposure (Donaghy et al 1989) or two different infections such as respiratory syncytial virus and campylobacter jejuni (Hayashi et al 1993) or booster vaccinations with tetanus toxoid[5, 48].

Relapsing forms of Guillain-Barré Syndrome can be distinguished from relapsing forms of CIDP by the rapidity of onset, the marked degree of recovery, normal CSF protein at the onset of an attack, the high incidence of preceding infections and the lack of response to immunosuppressant drugs (Grand'Maison et al 1992)[5].

The Guillain-Barré Syndrome Association of New South Wales is a registered charity that provides information and support to those affected by Guillain-Barré Syndrome, CIDP, and related conditions.

MILLER-FISHER SYNDROME

The Miller-Fisher syndrome which accounts for 5% of Guillain-Barré Syndrome cases[25, 32], is characterized by ophthalmoplegia, ataxia and areflexia. Patients present with diplopia followed by gait and limb ataxia. Ocular signs range from complete ophthalmoplegia, including uncreative pupils, to external opthalmoparesis with or without ptosis. Cranial nerves other than ocular motor nerves may be affected[31].

Motor strength is characteristically preserved[21]. The ataxia is attributed to a peripheral mismatch between propriceptive input from muscle spindles and kinesthetic information from joints receptors[48]. Patients presents with rapid onset of symmetrical, multiple cranial nerve palsies, most notably bilateral facial palsy (polyneuritis cranialis) [42], that may be a forme fruste of this syndrome.

Electrodiagnostic studies demonstrate an axonal process affecting predominantly sensory fibres with only mild motor conduction abnormalities[32]. Sensory nerve action potential amplitudes are usually normal.

MFS has a benign prognosis, with recovery after a mean of 10 weeks[26].

FUTURE THERAPIES[28, 38, 42]

Immunomodulatory treatment in the future may utilize recombinant cytokines (eg. Interlenkin-10, tumor growth factor, interferon-beta) and antibodies to inflammatory cytokines (eg. Tumor necrosis factor-alpha, soluble cytokine receptors or cytokine antagonist).

Treatment with P2 protein peptide 57-81, by nasal route has been found to be effective in rat experimental allergic neuritis. Sodium Fusidate which suppresses interlenkin-2, interferon and tumor necrosis factor-alpha both in vitro and in vivo also holds promise.

MATERIALS AND METHODS

Guillain-Barré Syndrome is a monophasic illness; often it is self-limiting. The initial assessment was based on clinical history, detailed neurological examination, routine investigations and special investigations like cerebrospinal fluid analysis and electrodiagnostic studies.

SELECTION OF PATIENTS

INCLUSION CRITERIA

1. Any patient admitted with features suggestive of flaccid progressive weakness affecting all the four limbs were included
2. Any patient admitted with progression of weakness of less than 4 weeks duration were included.
3. Any patient admitted with reduced or absent deep tendon reflexes were included

EXCLUSION CRITERIA

1. Any patient admitted with features of hypokalemic periodic paralysis.
2. Any patient admitted with features of upper motor neuron signs and symptoms
3. Any patient admitted with severe protopathic sensory symptoms
4. Any patient admitted with history of bite preceding the illness
5. Any patient admitted with history of exposure to toxins like organophosphates

6. Any patient with severe terminal illness

7. Patients admitted with history of suspected food poisoning

8. Patients in whom the weakness progressed for more than 4 weeks

Number of cases studied: 50

Duration of study: December 2005 – January 2007

Detailed neurological examination including higher mental functions, cranial nerves, motor system, sensory system and autonomic system was done for all 50 patients. Motor power in these patients were assessed according to Medical Research Council grading.

Autonomic dysfunction was looked for in all these patients. History of dryness of mouth, postural giddiness and defective sweating over the body were specifically asked for.

Blood pressure was routinely taken in lying and sitting posture and if possible in standing posture to bring out orthostatic hypotension. Sympathetic skin response was not done due to technical problem.

Respiratory function tests were done in all patients, everyday during hospitalization, including breath-holding time, single breath count, blowing candle at one arm length, chest expansion, Litten's phenomenon.

Likewise, basic investigations like complete blood count, peripheral smear, blood sugar and urea, serum creatinine and electrolytes, erythrocyte sedimentation rate, daily electrocardiogram, chest x-ray were done for all the 50 patients.

Lumbar puncture was done for 42 patients and CerebroSpinal Fluid was sent for Gram's stain, biochemical and cytological analysis.

Electrophysiological studies were conducted by using the machine RMS ADVANCE TESTING LAB. Nerve conduction studies were done in both upper and lower limbs.

In upper limbs, proximal latency, distal latency, motor nerve conduction velocity, F-response were studied in ulnar, median and radial nerves.

In lower limbs, similarly proximal latency, distal latency, motor nerve conduction velocity, F-response, H -reflex were studied in sciatic, lateral popliteal and posterior tibial nerves.

Sensory conduction velocity were studied in median nerve, ulnar nerve and sural nerve.

Electromyography was done with surface electrodes in thenar and hypothenar muscles, quadriceps, calf muscles, extensor digitorum.

Insertional activity was recorded.

Resting activity was recorded. Fibrillation potential, fasciculation potential, positive sharp waves were looked for.

Recruitment and interference pattern were looked for.

Compound muscle action potential was recorded.

Magnetic Resonance Imaging was done in 4 patients who presented with altered sensorium, sensory disturbance and urinary retention.

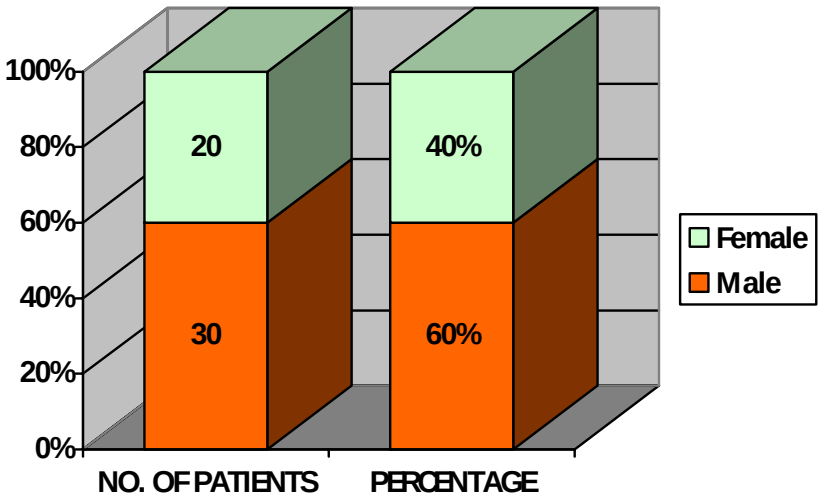
OBSERVATION AND RESULTS

SEX INCIDENCE

In the 50 patients studied, 30 were male and 20 were female.

TABLE 1

SEX	NO. OF PATIENTS	PERCENTAGE
Male	30	60%
Female	20	40%

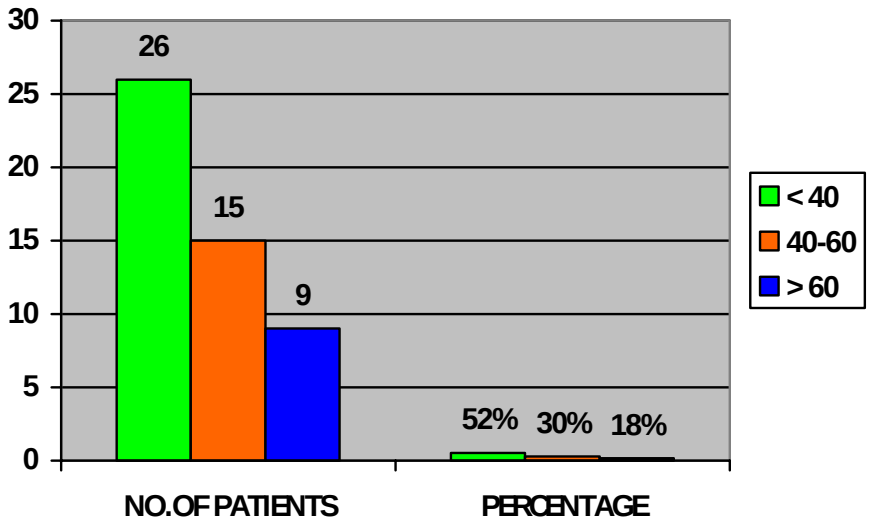


AGE INCIDENCE

All the 50 patients were above the age of 15; among which 26 (52%) patients were below the age of 40, 15 patients (30%) were between 40-60years, 9 patients (18%) were above the age of 60.

TABLE 2

AGE IN YEARS	NO.OF PATIENTS	PERCENTAGE
< 40	26	52%
40-60	15	30%
> 60	9	18%

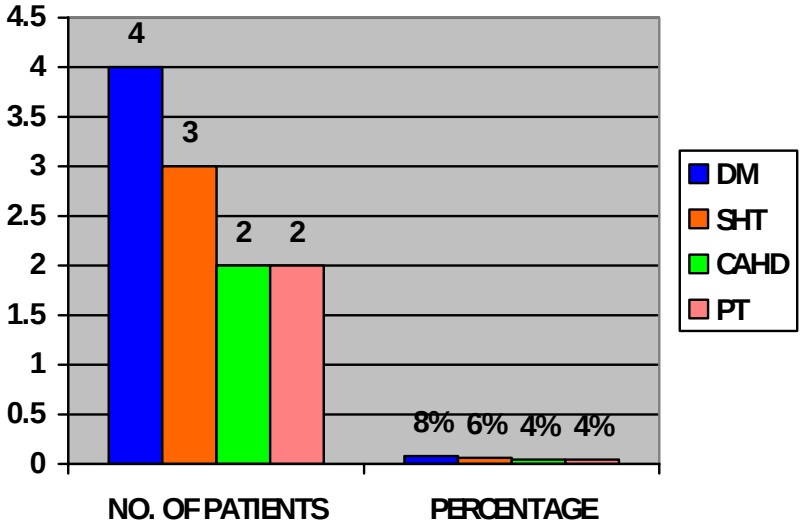


CO-EXISTING MEDICAL ILLNESS

4 patients were on treatment for diabetes mellitus, 3 patients were on treatment for hypertension, 2 patients were on treatment for pulmonary tuberculosis and ischemic heart disease.

TABLE 3

Sl.No	ILLNESS	NO. OF PATIENTS	PERCENTAGE
1	DM	4	8%
2	SHT	3	6%
3	CAHD	2	4%
4	PT	2	4%



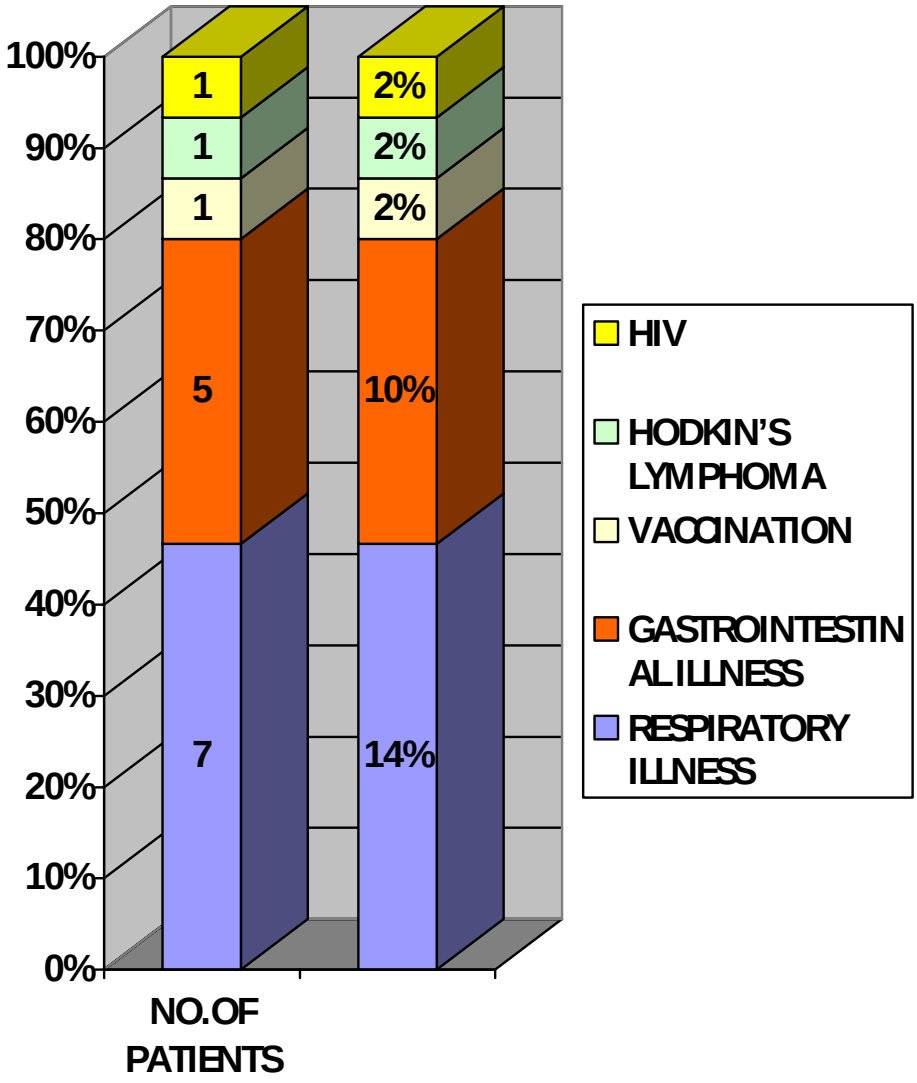
ANTECEDENT EVENTS

7 patients gave history of upper respiratory tract infection preceding the neurological illness. 5 patients gave history of gastroenteritis preceding the illness. 1 patient gave history of vaccination for dog bite preceding the illness.

1 patient was reactive for HIV. 1 patient was found to have generalised lymphadenopathy and mild hepatosplenomegaly, and Fine Needle Aspiration Cytology proved to be Hodgkin's lymphoma.

TABLE 4

Sl.No.	ANTECEDENT EVENTS	NO.OF PATIENTS	PERCENTAGE
1	RESPIRATORY ILLNESS	7	14%
2	GASTROINTESTINAL ILLNESS	5	10%
3	VACCINATION	1	2%
4	HODKIN'S LYMPHOMA	1	2%
5	HIV	1	2%


100%
90%
80%
70%
60%
50%
40%
30%
20%
10%
0%
1
1
1
5
7
2%
2%
2%
10%
14%
HIV
HODKIN'S LYMPHOMA
VACCINATION
GASTROINTESTINAL ILLNESS
RESPIRATORY ILLNESS
NO.OF PATIENTS

CLINICAL PRESENTATION

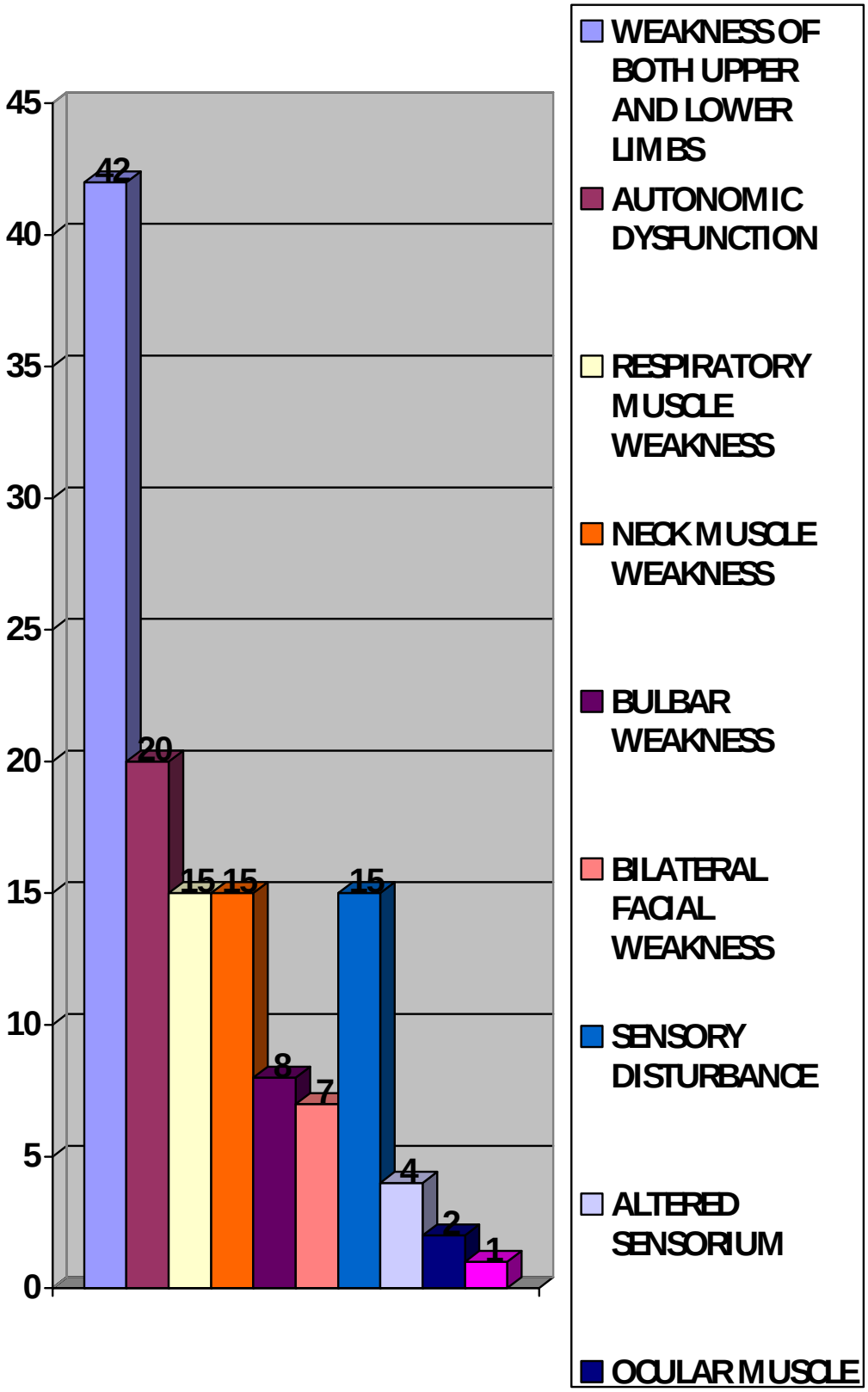
In 50 patients studied, 42 patients had weakness of both upper and lower limbs; 15 patients had neck muscle weakness; 8 patients had features suggestive of bulbar weakness; 2 patients had ocular muscle involvement.

15 patients gave history suggestive of sensory disturbance and 12 patients had electrophysiological evidence of sensory involvement. 7 patients had bilateral lower motor neuron type of facial weakness.

4 patients developed altered sensorium for a short period with complete recovery within days.

TABLE 5

Sl. No.	CLINICAL PRESENTATION	NO.OF PATIENTS	PERCENTAGE
1	WEAKNESS OF BOTH UPPER AND LOWER LIMBS	42	84%
2	AUTONOMIC DYSFUNCTION	20	40%
3	RESPIRATORY MUSCLE WEAKNESS	15	30%
4	NECK MUSCLE WEAKNESS	15	30%
5	BULBAR WEAKNESS	8	16%
6	BILATERAL FACIAL WEAKNESS	7	14%
7	SENSORY DISTURBANCE	15	30%
8	ALTERED SENSORIUM	4	8%
9	OCULAR MUSCLE INVOLVEMENT	2	4%
10	ATAXIA	1	2%


45
40
35
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25
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42
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4
2
1
WEAKNESS OF BOTH UPPER AND LOWER LIMBS
AUTONOMIC DYSFUNCTION
RESPIRATORY MUSCLE WEAKNESS
NECK MUSCLE WEAKNESS
BULBAR WEAKNESS
BILATERAL FACIAL WEAKNESS
SENSORY DISTURBANCE
ALTERED SENSORIUM
OCULAR MUSCLE

Among these 50 patients, 15 patients developed respiratory distress in some form and 5 of them were provided ventilator for life support. 3 out of the 5 patients on ventilator died without improvement. Remaining 2 patients were weaned from ventilator after 4 days with favorable outcome.

2 patients died due to respiratory distress, before giving ventilatory support. 3 patients who developed severe respiratory distress were discharged as against medical advice and follow up was not recorded.

20 patients presented with some form of autonomic dysfunction. Orthostatic hypotension was found in 8 patients. Cardiac arrhythmias in electrocardiogram including ventricular, atrial ectopics, sinus tachycardia and ST-T changes were noted in 7 patients. Remaining 5 patients had features of bladder disturbance in the form of urinary retention in whom catheterisation was done.

Among 15 patients who had respiratory distress, 11 patients showed autonomic disturbance. All patients who expired and who were on ventilator had autonomic disturbance.

CEREBROSPINAL FLUID ANALYSIS

Cerebrospinal fluid analysis was done for 42 patients. Cerebrospinal fluid protein was increased in all the 42 patients ranging from 260mgs% to 1.5gms%. Cerebrospinal fluid cell count was within normal limits, i.e. less than 50 mononuclear cells per cubic millimeter. Cell count was 250 in one patient with HIV infection.

TABLE 6.1

CELL COUNT per cubic mm	< 10	10-20	21-30	31-40	41-50	>50
NO.OF PATIENTS	5	9	19	7	1	1

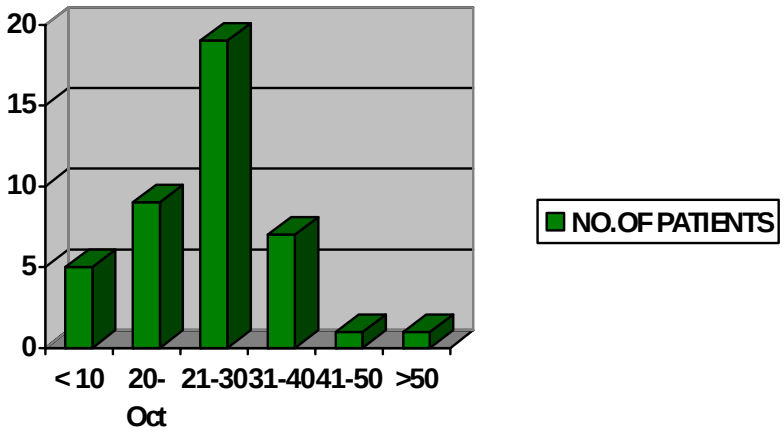
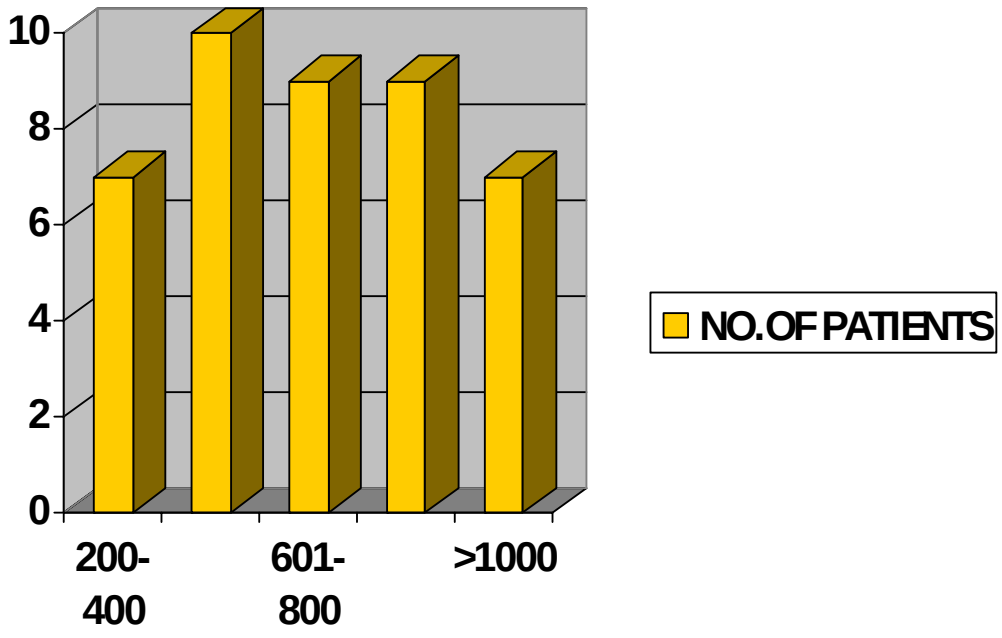


TABLE 6.2

PROTEIN (mgs %)	200-400	401-600	601-800	800-1000	>1000
NO.OF PATIENTS	7	10	9	9	7



All these 50 patients had abnormal electrodiagnostic studies like

1. Motor Nerve Conduction Velocity was decreased in all the 4 limbs varying from mild to severe degree.

2. Conduction block was present in 6 patients.

3. H- reflex could not be elicited in all the patients.

4. F- response was prolonged in 8 patients and absent in 42 patients.

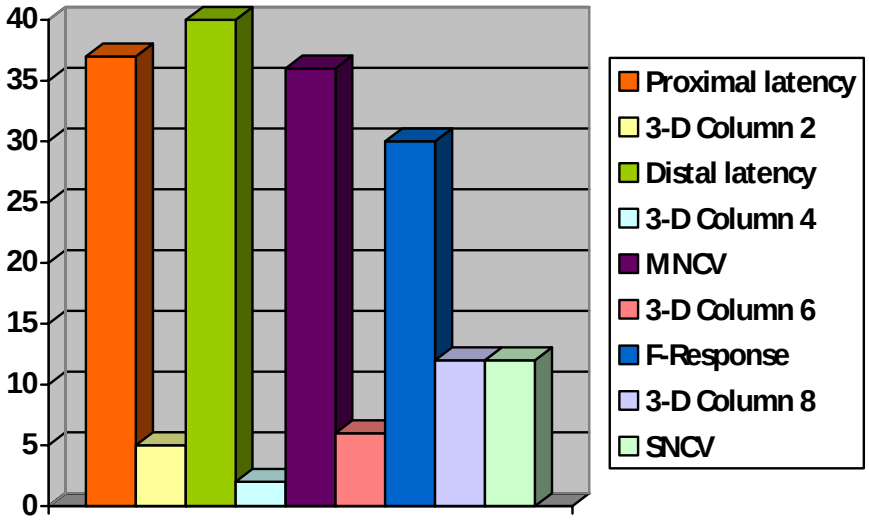
5. Compound Muscle Action Potential was decreased in more than 40 patients.

ELECTRODIAGNOSTIC STUDIES

NERVE CONDUCTION STUDIES UPPER LIMBS

TABLE 7

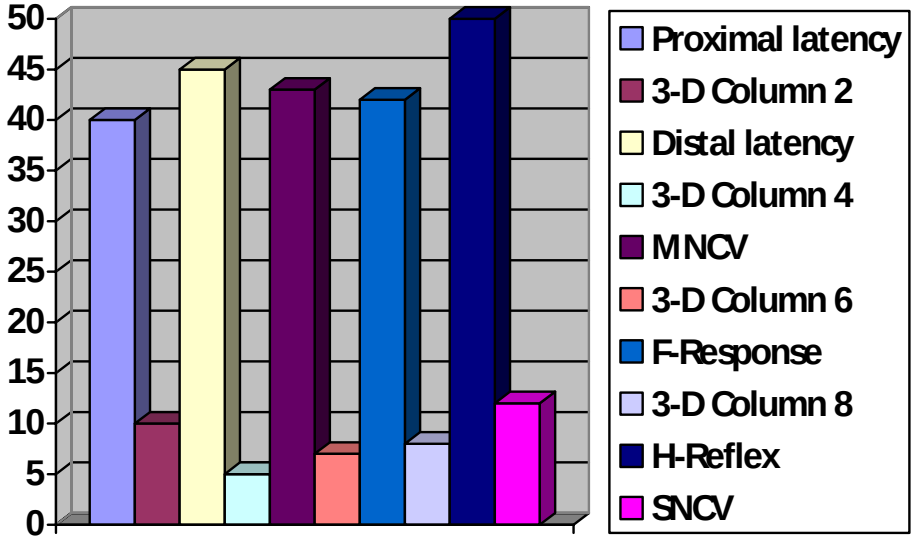
SL. NO		NO. OF PATIENTS	ULNAR	MEDIAN	RADIAL
1	Proximal latency	37	Prolonged	Prolonged	Prolonged
		5	Normal	Normal	Normal
2	Distal latency	40	Prolonged	Prolonged	Prolonged
		2	Normal	Normal	Normal
3	MNCV	36	Delayed	Delayed	Delayed
		6	Normal	Normal	Normal
4	F-Response	30	Absent	Absent	Absent
		12	Prolonged	Prolonged	Prolonged
5	SNCV	12	Absent	Absent	-



NERVE CONDUCTION STUDIES LOWER LIMBS

TABLE 8

SL. NO		NO. OF PATIENTS	COMMON PERONEAL	POSTERIOR TIBIAL	SURAL
1	Proximal latency	40	Prolonged	Prolonged	-
		10	Normal	Normal	
2	Distal latency	45	Prolonged	Prolonged	-
		5	Normal	Normal	
3	MNCV	43	Delayed	Delayed	-
		7	Normal	Normal	
4	F-Response	42	Absent	Absent	-
		8	Prolonged	Prolonged	
5	H-Reflex	50	Absent	Absent	-
6	SNCV	12	-	-	Absent



The disease progressed in 40 patients upto 14 days. 35 patients worsened in the first week. 15 patients worsened after the first week.

40 patients had shown some improvement during hospital stay itself. 10 patients did not show any improvement during hospital stay. Among 40 patients in whom improvement had seen, recovery was rapid during the first week in 35 patients.

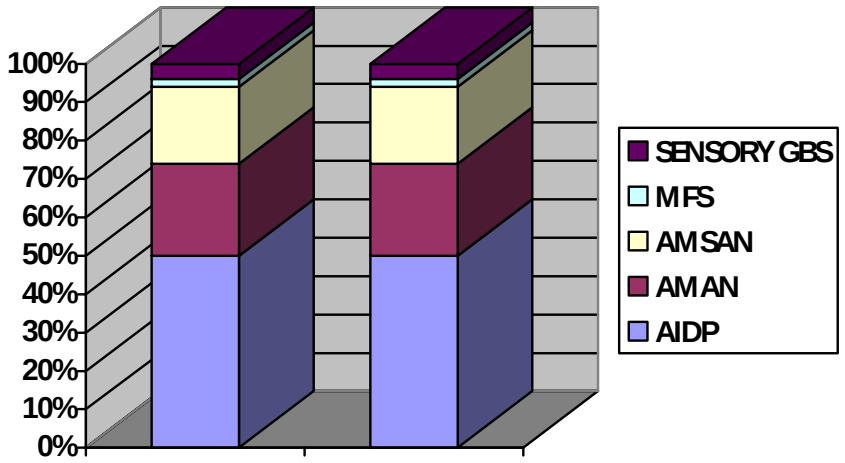
Cerebrospinal fluid analysis and electrodiagnostic studies were done only once during their hospital stay and follow up study could not be done.

Magnetic resonance imaging was done in 4 patients which showed no evidence for cord compression or myelitis.

TYPES OF GUILLAIN-BARRÉ SYNDROME

TABLE 10

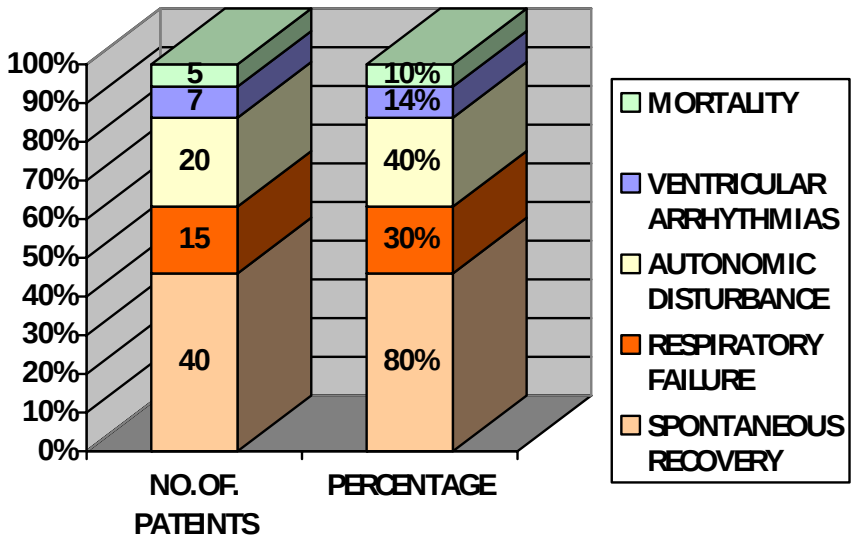
S.NO	TYPES	NO.OF PATIENTS	PERCENTAGE
1.	AIDP	25	50%
2.	AMAN	12	24%
3.	AMSAN	10	20%
4.	MFS	1	2%
5.	SENSORY GBS	2	4%



OUTCOME OF PATIENTS

TABLE 11

S.NO	OUTCOME	NO.OF. PATEINTS	PERCENTAGE
1	SPONTANEOUS RECOVERY	40	80%
2	RESPIRATORY FAILURE	15	30%
3	AUTONOMIC DISTURBANCE	20	40%
4	VENTRICULAR ARRHYTHMIAS	7	14%
5	MORTALITY	5	10%



DISCUSSION

50 patients were included in our study. The incidence of Guillain-Barré Syndrome was around 1% in total hospital admissions in neurology ward.

Sex prevalence in our study was 60% (30) for men and 40% (20) for women. Seasonal variation was observed in our study, between July and October. This observation is supported by reports from India and China.

Among the 50 patients studied, 52 % (26) of patients were below 40 years, 30% (15) of patients were between 40-60 years and 18% (9) of patients were above the age of 60. The age range of our consecutive patients has been 16 years to 78 years, with attack rates highest in persons less than 50 years of age.

Those patients with coexisting illness like diabetes mellitus, systemic hypertension, ischemic heart disease and pulmonary tuberculosis were above the age of 40 and they had delayed recovery. Approximately one third of patients reported a history of an antecedent event. According to the study by Rees et al, 1995 preceding diarrhoeal illness, especially Campylobacter jejuni infection was documented in cases of acute motor axonal neuropathy; supporting our study.

30% (15) of patients developed respiratory muscle weakness in some form. Diaphragmatic movement was assessed by Litten's phenomenon. Diaphragmatic weakness was increased during the second week and began to recover after the third week.

In patients with respiratory distress, need for ventilatory support was assessed by respiratory rate, single breath count, chest expansion, oxygen saturation and appearance of central cyanosis. In a study by Sharshar et at, 2003, short disease duration, inability to lift the head, and a vital capacity of less than 60% predicted the need for mechanical ventilation in 85% of patients. Accordingly, 10% (5) of our patients were put on ventilator.

Autonomic dysfunction was observed in 40% (20) of patients. Orthostatic hypotension was detected in 8 patients; Abnormal sweating either increased or decreased sweating was looked for in the trunk and limbs. Arrhythmias were found in 7 patients. Benign arrhythmias like ventricular and atrial ectopics, and non specific ST-T changes, sinus tachycardia, less often sinus bradycardia were noted in 5 patients, which persisted for less than a week. Fatal ventricular arrhythmias were found in 2 patients.

In a study by Zochodne et al in 1994, autonomic dysfunction was observed in 65% of patients and in another study by Winner and Hughes in 1998, cardiac arrhythmias due to autonomic dysfunction was the leading cause of death, which

contributed by 7 percent. As per the above study major causes of death in our study were due to respiratory failure and cardiac arrhythmias.

Most of the patients with respiratory failure were found to have autonomic dysfunction and recovery was delayed. Mortality was increased in patients who have combined respiratory failure on ventilatory support and autonomic dysfunction, especially orthostatic hypotension and ventricular arrhythmias.

Bladder disturbance was one of the clinical presentations in 5 patients, all of them were above the age of 60 for whom catheterisation was not needed for more than a week.

15 patients had sensory disturbance in the form of impaired position and vibration sense. Study by Winner et al in 1998 support this study. 12 patients were found to have sensory involvement on electrodiagnostic studies.

Most of the studies do not recommend the use of steroids. Yet many centers use high dose oral prednisolone or methylprednisolone. In our hospital, we do not use steroids routinely, some patients included in our study, had been treated with steroids outside.

6% (3) of patients on steroids developed peptic ulceration and gastrointestinal bleeding, with delayed recovery.

2% (1) of patients with diabetes mellitus who were put on steroids developed ketoacidosis and turned out with delayed recovery.

In our study, there was no significant difference in the outcome of patients treated with or without steroids, similar to the word literature. A randomized trial of oral prednisolone therapy by Guillain-Barré Syndrome steroid trial group in 1993, showed no benefit. A study by Hughes et al 1978, suggested that steroids might increase the subsequent relapse rate.

Those patients above the age of 60 without any medical illness also, showed delayed recovery. The North American Guillain-Barré Syndrome study group support our study.

4% (2) of the patients presented with ocular muscle involvement; 2% (1) of the patients had ataxia, with favorable outcome.

Prognosis in patients with Guillain-Barré Syndrome varied linearly with severity of demyelination or axonal degeneration detected by electrodiagnostic studies.

Recovery was delayed in patients with conduction block than in patients with delayed motor nerve conduction velocity alone.

Recovery was earlier and favorable in patients with absent H-reflex and F-response and delayed motor conduction than in patients with conduction block. A distal CMAP amplitude of less than 20% of the lower limits of normal was associated with poor outcome in the North American Guillain-Barré syndrome study group.

In our study, mortality was around 10 percent. Commonest cause of mortality were respiratory failure and ventricular arrhythmias.

Patients with high protein content in cerebrospinal fluid indicating demyelination of nerve roots showed delayed recovery.

In a study by Ropper and Marmarou, the increase in cerebrospinal fluid protein had no clinical or prognostic significance.

CONCLUSION

1. The incidence of Guillain-Barré Syndrome in our study was around 1% among total hospital admissions in neurology ward.

2. There was no significant sex preponderance in our study.

3. 80% of Guillain-Barré Syndrome patients recovered smoothly without going for complications.

4. 10% of patients developed bulbar weakness of varying severity.

5. 30% of patients developed neck muscle weakness of varying severity.

6. 30% of Guillain-Barré Syndrome patients developed respiratory muscle weakness of varying severity.

7. 10% of patients needed ventilatory support to maintain oxygen saturation.

8. 40% of patients showed features of autonomic disturbance of varying severity.

9. 2 patients had features suggestive of ocular muscle involvement and in 1 patient features of incordination was present.

10. Prognostic outcome in our study is somewhat poor with increasing age.

11. Prognostic outcome is poor when there is co-existing illness like diabetes mellitus or ischemic heart disease.

12. Cerebrospinal fluid analysis in patients the Guillain-Barré Syndrome who had increased protein correlated with severe demyelination in electrodiagnostic studies and delayed recovery

13. Prognosis in patients the Guillain-Barré Syndrome linearly varies with severity of electrodiagnostic studies.

    H-reflex was invariably absent in all those patients included in the study.

    F- response was absent in 90% of patients in lower limbs and 70% of patients in upper limbs.

    Reduction in motor nerve conduction velocity was noted in all these patients of varying severity.

    A feature of conduction block was noted in patients with severe weakness.

    Recovery was delayed in patients with conduction block than in patients with delayed motor nerve conduction velocity alone.

14. In our study, there was no significant difference in the outcome between patients treated with or without steroids.

15. Mortality was around 10% in our study.

16. The commonest cause of mortality was respiratory failure and fatal ventricular arrhythmias.

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PROFORMA

Name **Age / sex** **IP No.**

Place **Occupation**

History

Duration of illness

Progression of symptoms

Motor symptoms

Sensory symptoms

Autonomic symptoms

H/o respiratory distress

Preceding respiratory or gastrointestinal illness

H/o vaccination

Any other relevant history

Examination

Higher mental functions

Cranial nerves

Spinomotor system

Nutrition

Tone

Power

Reflexes

Neck muscle weakness

Sensory system

Autonomic system

Orthostatic hypotension

Sympathetic skin function

Sudomotor

Secretomotor

Pilomotor

Gait

Other systems

RS

Chest expansion

Single breath count

Candle blow time

Breath holding time

CVS

ABD

Investigations

Blood CBC

Biochemistry

ECG

Chest X-Ray PA view

Pulmonary function tests

CSF analysis

Electrophysiological studies

EMG

CMAP

Interference

Recruitment

Resting activity

Insertional activity

Nerve conduction studies

Motor

UL – median

radial

ulnar

LL - posterior tibial

Common peroneal

Sensory

UL - median

Ulnar

LL - sural

Late responses

F wave

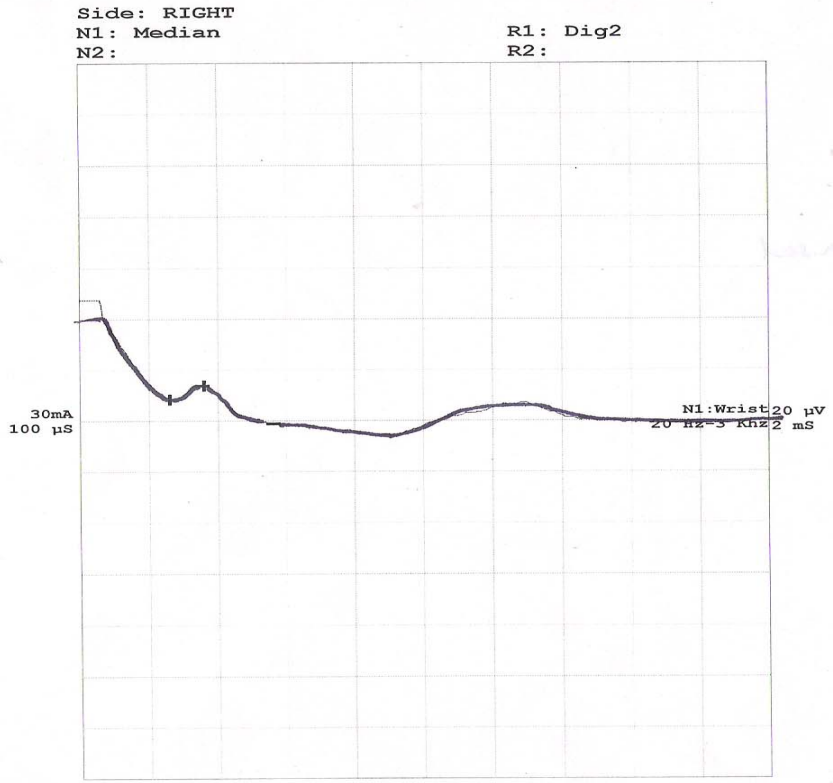
H reflex

RMS ADVANCE TESTING LAB

181/5 PHASE I INDUSTRIAL AREA CHANDIAGARH

PHONES 658701-705

Mr. Prakash **35/M** **SNC RECORD**



Stim Site	Lat1 (mS)	Lat2 (mS)	Dur (mS)	Amp	Area
Wrist	02.58	03.58	01.00	15.1 µV	02.7 µVmS

Segment	Diff (mS)	Dist (mm)	NCV (m/S)
Dig2 - Wrist	02.58	150	58.14

Rt. Median - Amplitude decreased

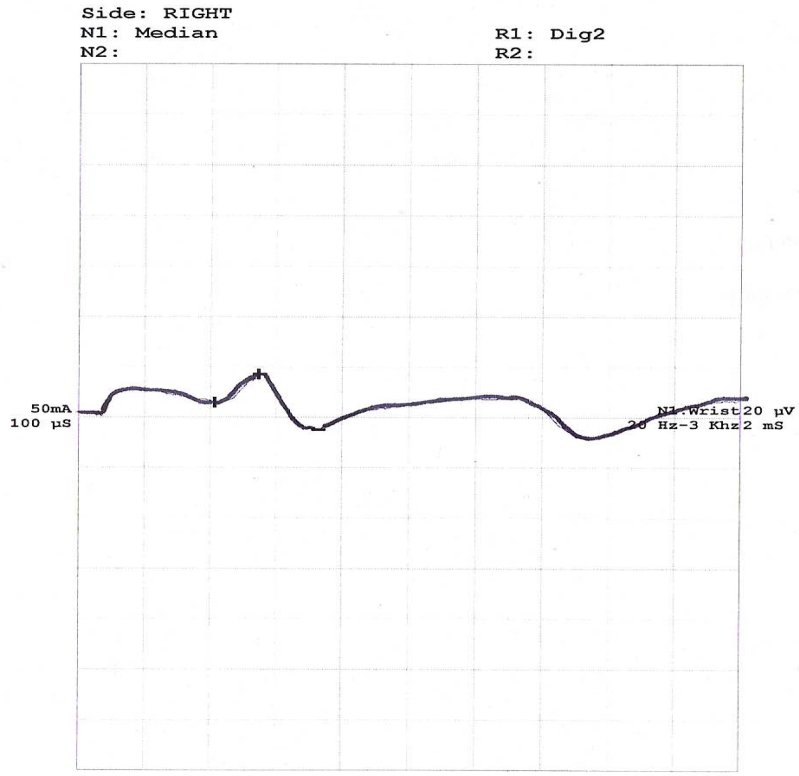
Test Comments

RMS ADVANCE TESTING LAB

181/5 PHASE I INDUSTRIAL AREA CHANDIAGARH

PHONES 658701-705

Mr. Krishnamoorthy **64/M** **SNC RECORD**



Stim Site	Lat1 (mS)	Lat2 (mS)	Dur (mS)	Amp	Area
Wrist	04.13	05.46	01.33	22.1 µV	05.8 µVmS

Segment	Diff (mS)	Dist (mm)	NCV (m/S)
Dig2 - Wrist	04.13	160	38.83

Rt. Median - Amplitude- Decreased
Latency prolonged

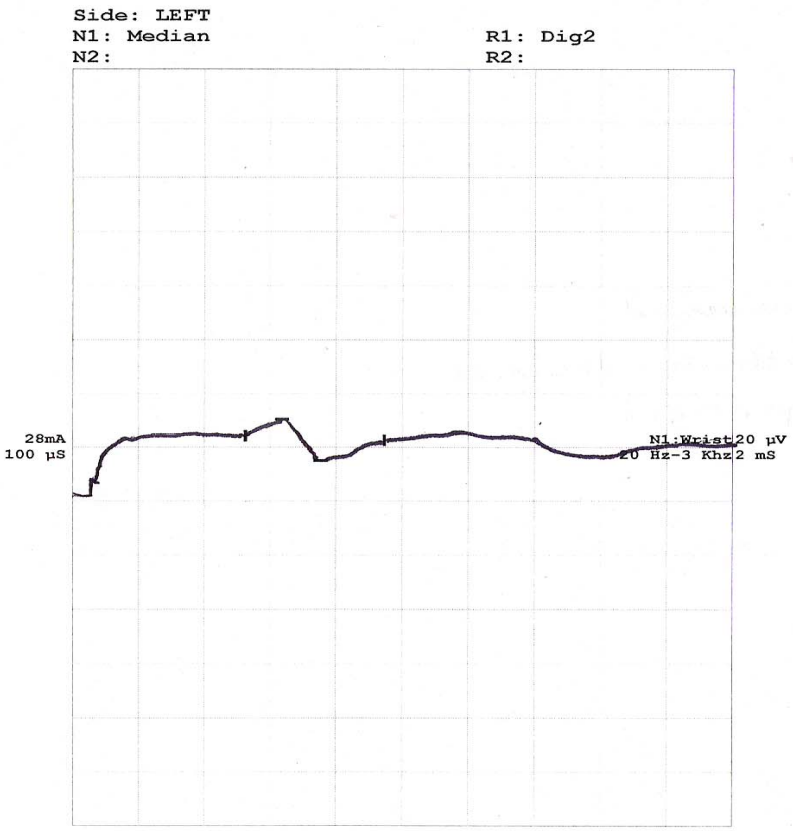
Test Comments

RMS ADVANCE TESTING LAB

181/5 PHASE I INDUSTRIAL AREA CHANDIAGARH

PHONES 658701-705

Mr. Shanmugam **48/M** **SNC RECORD**



Stim Site	Lat1 (mS)	Lat2 (mS)	Dur (mS)	Amp	Area
Wrist	05.25	09.46	04.21	15.4 µV	19.5 µVmS

Segment	Diff (mS)	Dist (mm)	NCV (m/S)
Dig2 - Wrist	05.25	150	28.57

Lt. Median
Amplitude - decreased
Conduction velocity- decreased
latency prolonged.

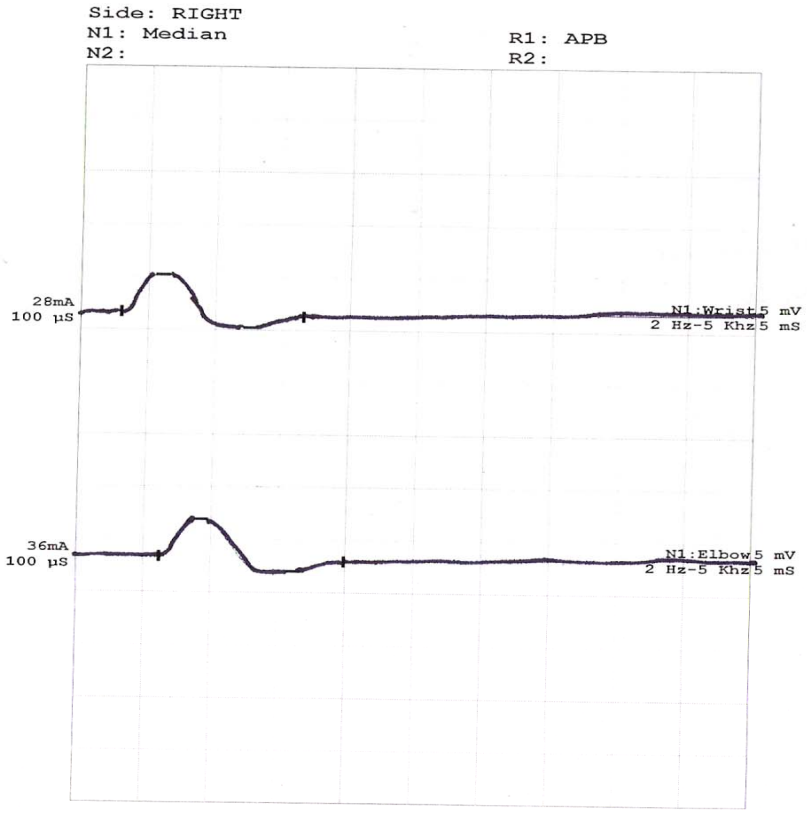
Test Comments

RMS ADVANCE TESTING LAB

181/5 PHASE I INDUSTRIAL AREA CHANDIAGARH

PHONES 658701-705

Mrs. Maheswari **35/F** **MNC RECORD**



Stim Site	Lat1 (mS)	Lat2 (mS)	Dur (mS)	Amp	Area
Wrist	03.02	16.56	13.54	05.1 mV	20.5 mVmS
Elbow	06.15	19.90	13.75	04.8 mV	19.7 mVmS

Segment	Diff (mS)	Dist (mm)	NCV (m/S)
Wrist - Elbow	03.13	170	54.31

Rt. Median - Amplitude decreased
CMAP - Prolonged

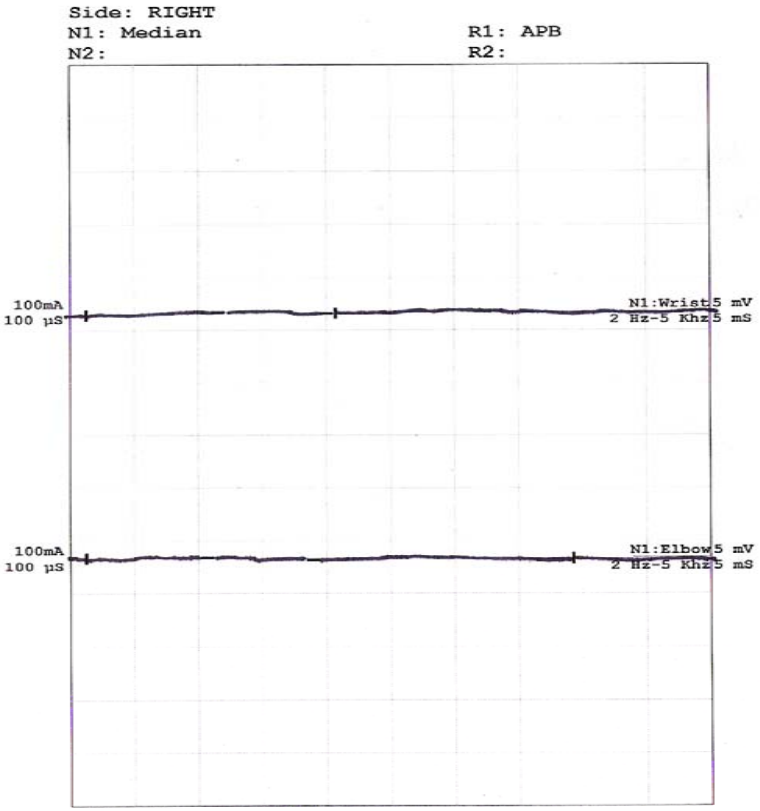
Test Comments

RMS ADVANCE TESTING LAB

181/5 PHASE I INDUSTRIAL AREA CHANDIAGARH

PHONES 658701-705

Mrs. Dhanabakkiyam **46/F** **MNC RECORD**



Stim Site	Lat1 (mS)	Lat2 (mS)	Dur (mS)	Amp	Area
Wrist	01.25	20.73	19.48	154.9 µV	4409.4 µVmS
Elbow	01.25	39.27	38.02	216.0 µV	2402.8 µVmS

Segment	Diff (mS)	Dist (mm)	NCV (m/S)
Wrist - Elbow	00.00		

Rt. Median - Mixed Demyelinating and axonal pattern

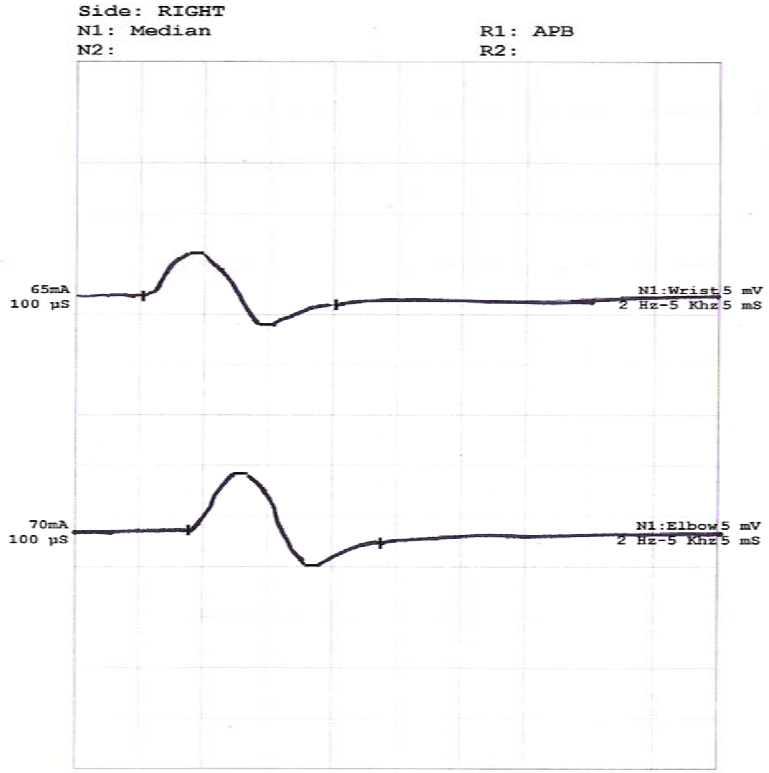
Test Comments

RMS ADVANCE TESTING LAB

181/5 PHASE I INDUSTRIAL AREA CHANDIAGARH

PHONES 658701-705

Mrs. Alamelu **62/F** **MNC RECORD**



Stim Site	Lat1 (mS)	Lat2 (mS)	Dur (mS)	Amp	Area
Wrist	05.21	20.21	15.00	07.1 mV	32.1 mVmS
Elbow	08.85	23.75	14.90	09.0 mV	41.1 mVmS

Segment	Diff (mS)	Dist (mm)	NCV (m/S)
Wrist - Elbow	03.64	170	46.70

Rt. Median - Amplitude decreased
Distal latency Prolonged

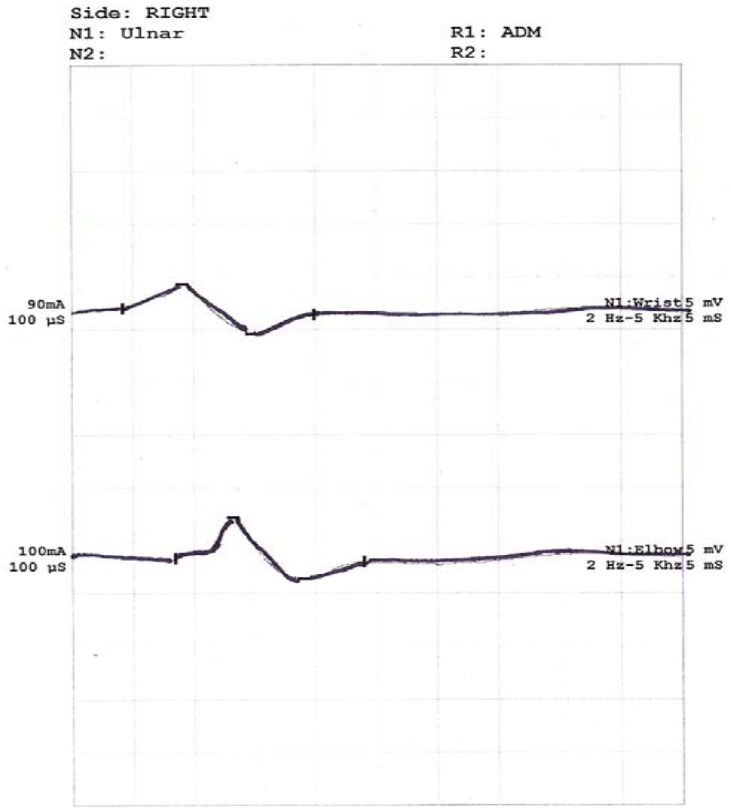
Test Comments

RMS ADVANCE TESTING LAB

181/5 PHASE I INDUSTRIAL AREA CHANDIAGARH

PHONES 658701-705

Mr. Munuswamy **65/M** **MNC RECORD**



Stim Site	Lat1 (mS)	Lat2 (mS)	Dur (mS)	Amp	Area
Wrist	04.17	19.90	15.73	04.6 mV	19.8 mVmS
Elbow	08.44	23.96	15.52	05.9 mV	21.0 mVmS

Segment	Diff (mS)	Dist (mm)	NCV (m/S)
Wrist - Elbow	04.27	210	49.18

Rt. ulnar - Amplitude decreased
Conduction Velocity - Prolonged
Temporal dispersion

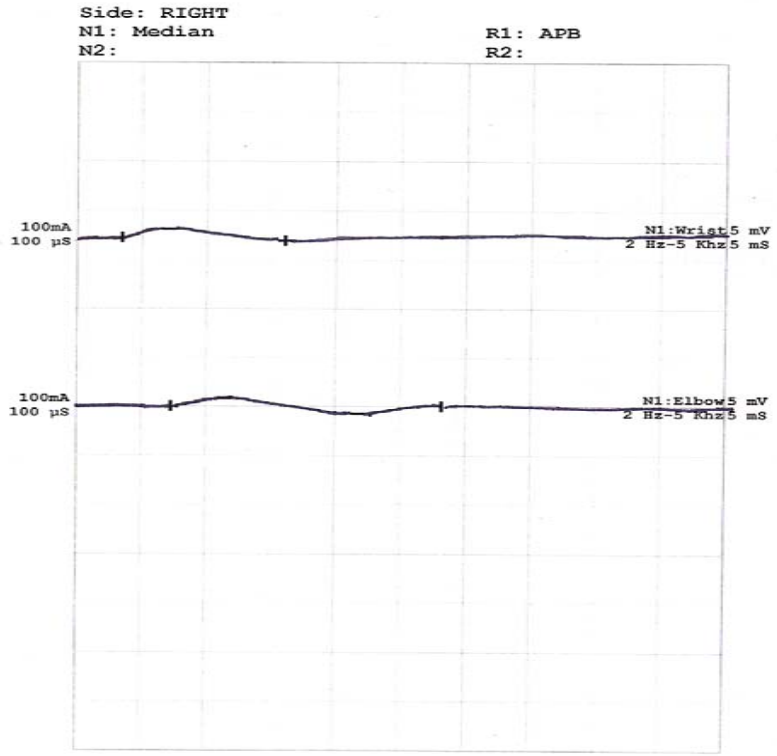
Test Comments

RMS ADVANCE TESTING LAB

181/5 PHASE I INDUSTRIAL AREA CHANDIAGARH

PHONES 658701-705

Mr. Alexander **25/M** **MNC RECORD**



Stim Site	Lat1 (mS)	Lat2 (mS)	Dur (mS)	Amp	Area
Wrist	03.44	16.04	12.60	01.2 mV	05.1 mVmS
Elbow	07.29	28.13	20.83	01.5 mV	08.4 mVmS
Axilla					

Segment	Diff (mS)	Dist (mm)	NCV (m/S)
Wrist - Elbow	03.85	200	51.95
Elbow - Axilla			
Elbow - Axilla			

Rt. Median - Amplitude - decreased
Temporal dispersion

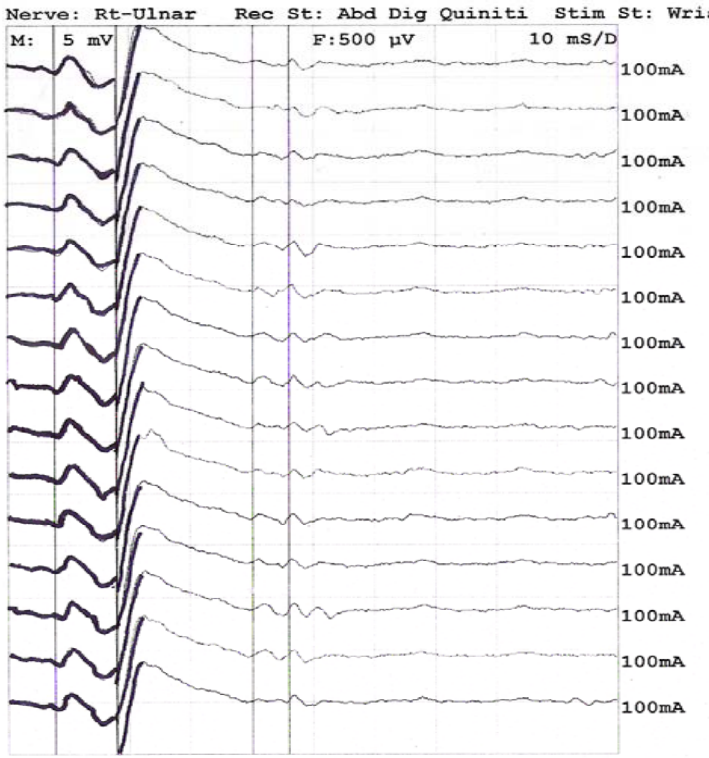
Test Comments

RMS ADVANCE TESTING LAB

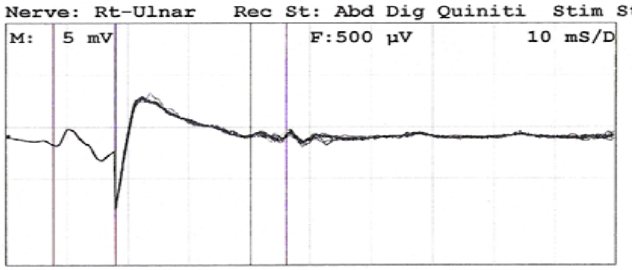
181/5 PHASE I INDUSTRIAL AREA CHANDIAGARH

PHONES 658701-705

Mr. Ponnusamy **61/M** **F-WAVE RECORD**



M Lat	Fmin Lat	Fmax Lat	Fmean Lat	(F-M) Lat	Distance	Velocity
7.7 mS	40.2 mS	46.0 mS	43.1 mS	32.5 mS	00 mm	00.0 m/S



Rt. Ulnar - F-Response Prolonged

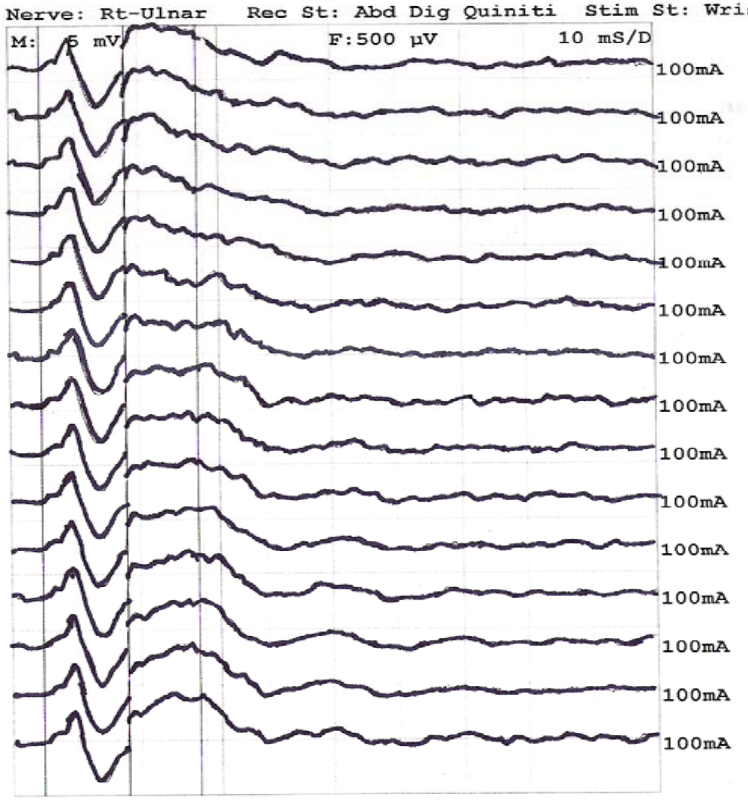
Test Comments

RMS ADVANCE TESTING LAB

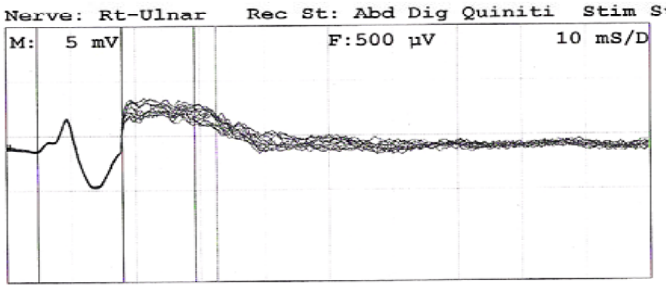
181/5 PHASE I INDUSTRIAL AREA CHANDIAGARH

PHONES 658701-705

Mr. Chidambaram **55/M** **F-WAVE RECORD**



M Lat	Fmin Lat	Fmax Lat	Fmean Lat	(F-M) Lat	Distance	Velocity
4.8 mS	29.2 mS	32.5 mS	30.8 mS	24.4 mS	00 mm	00.0 m/S



Rt. Ulanar - F-response absent

Test Comments

S.No.	Name	Age /Sex	IP.NO	Clinical Presentation	Antecedent events	Co-existing Medical illness	Autonomic dysfunction(including ECG)	Serum potassium (meq/Lt)	CSF analysis		MRI	EMG Types	Final outcome
									Protein mgs%	Cell count			
1	Govindaraj	33/M	838282	Weakness of UL, LL, neck muscles	-	-	-	3.8	260	30	-	AIDP	Spontaneous recovery
2	Maruthamuthu	78/M	838304	Weakness of UL, LL, neck muscles, bulbar, respiratory muscles, sensory disturbance	GE	CAHD	Ventricular arrythmias, Urinary retention	4.1	-	-	-	AMAN	Expired
3	Alexander	25/M	838398	Weakness of UL, LL neck muscles	-	-	-	4.0	1340	35	-	AIDP	Spontaneous and delayed recovery
4	Renuka	23/F	838456	Weakness of UL, LL	-	-	Sinus Tachycardia	4.8	300	40	-	AMAN	Spontaneous recovery
5	Vadivelu	55/M	838821	Weakness of UL, LL	-	-	-	3.9	7280	20	-	AIDP	Spontaneous and delayed recovery
6	Arunachalam	66/M	839907	Weakness of UL, LL, neck , bulbar and respiratory muscles, sensory disturbance	GE	-	Ventricular ectopics	3.9	-	-	-	AIDP	Expired
7	Shanmugam	48/M	841031	Weakness of UL, LL, neck muscles, sensory disturbance	URTI	-	Orthostatic hypotension	3.8	420	25	-	AMSAN	Spontaneous recovery
8	Arul Victor	21/M	841099	Weakness of UL, LL	-	Hodgkin's lymphoma	-	4.2	1460	35	-	AIDP	Spontaneous and delayed recovery
9	Maheswari	35/F	843233	Weakness of UL, LL, neck muscles	-	T_2DM	Orthostatic hypotension	4.4	570	30	-	AMAN	Spontaneous recovery
10	Chidambaram	55/M	845312	Weakness of UL, LL, neck , bulbar and respiratory muscles	-	PT	Urinary retention	3.7	826	20	N	AIDP	Residual neurological deficit
11	Jothi	45/F	847210	Weakness of UL, LL	-	-	-	3.6	340	<10	-	AMAN	Spontaneous recovery
12	Ramaraj	67/M	847817	Weakness of UL, LL, neck muscles, sensory disturbance	URTI	-	Ventricular ectopics	3.7	750	28	-	AIDP	Spontaneous and delayed recovery
13	Rajeswari	32/F	848124	Weakness of UL, LL	-	-	-	4.0	340	20	-	AMAN	Spontaneous recovery
14	Parimala	63/F	849378	Weakness of UL, LL, neck, bulbar, ocular and respiratory muscles, sensory disturbance	GE	T_2DM	Orthostatic hypotension	3.5	-	-	-	AMSAN	Discharged as AMA
15	Kokilam	37/F	850014	Weakness of LL	-	-	-	4.3	600	<10	-	AMAN	Spontaneous and delayed recovery
16	Elangovan	51/M	8541812	Weakness of LL	-	-	-	4.3	430	25	-	AIDP	Spontaneous recovery
17	Banumathi	22/F	853321	Weakness of UL, LL, neck, bulbar and respiratory muscles	-	-	-	4.9	-	-	-	AIDP	Spontaneous recovery

S.No.	Name	Age /Sex	IP.NO	Clinical Presentation	Antecedent events	Co-existing Medical illness	Autonomic dysfunction(including ECG)	Serum potassium (meq/Lt)	CSF analysis		MRI	EMG Types	Final outcome
									Protein mgs%	Cell count			
18	Dhanalakshmi	26/F	855114	Weakness of UL, LL, neck muscles	-	-	Sinus tachycardia, urinary retention	4.2	705	25	N	AIDP	Spontaneous and delayed recovery
19	Renganathan	64/M	855999	sensory disturbance	-	-	Urinary retention	5.0	800	28	-	Sensory GBS	Spontaneous and delayed recovery
20	Vallarasan	16/M	856734	Weakness of UL, LL, neck muscles	-	-	-	4.7	430	15	-	AIDP	Spontaneous recovery
21	Selvam	52/M	858458	Weakness of UL, LL, neck, bulbar, and respiratory muscles, sensory disturbance	URTI	SHT/CAHD	Ventricular arrhythmias	4.2	327	25	-	AMSAN	Spontaneous recovery
22	Kalimuthu	50/M	858780	Weakness of LL	-	-	-	4.6	945	<10	-	AIDP	Spontaneous and delayed recovery
23	Muthuselvi	16/F	861325	Weakness of UL, LL,	-	-	-	4.5	600	36	-	AMAN	Spontaneous recovery
24	Prakash	35/M	863210	Weakness of UL, LL, neck muscles, sensory disturbance	HIV	-	-	4.6	1120	250	-	AMSAN	Expired
25	Manoj	30/M	864380	Weakness of UL, LL, neck, bulbar, and respiratory muscles,	URTI	-	-	5.0	810	22	-	AIDP	Spontaneous and delayed recovery
26	Chitra	23/F	867370	Weakness of UL, LL, neck muscles	-	-	-	3.5	725	20	-	AIDP	Spontaneous recovery
27	Ganesan	43/M	868721	Weakness of UL, LL, neck, bulbar, and respiratory muscles	-	-	-	3.7	-	-	-	AIDP	Discharged as AMA
28	Dhanabakkiyam	46/F	869529	Weakness of UL, LL, neck muscles, sensory disturbance	-	-	Orthostatic hypotension	3.7	608	26	-	AMSAN	Expired
29	Sathish	18/M	869636	Weakness of UL, LL	Vaccination for dog bite	-	-	4.1	300	<10	-	AMAN	Spontaneous recovery
30	Alamelu	62/F	871027	Weakness of UL, LL, neck muscles, sensory disturbance	-	-	Orthostatic hypotension	4.0	810	36	-	AMSAN	Spontaneous and delayed recovery
31	Ponnusamy	61/M	874109	Weakness of UL, LL, neck, bulbar, and respiratory muscles, sensory disturbance	GE	PT	Urinary retention	3.8	-	-	-	AMSAM	Discharged as AMA
32	Alamelu	16/F	874145	Weakness of UL, LL	-	-	Sinus Bradycardia	4.4	482	15	-	AMAN	Spontaneous recovery
33	Nithish Kumar	20/M	877611	Weakness of UL, LL, neck, bulbar, and respiratory muscles,	-	-	-	5.0	-	-	-	AIDP	Spontaneous recovery

S.No.	Name	Age /Sex	IP.NO	Clinical Presentation	Antecedent events	Co-existing Medical illness	Autonomic dysfunction(including ECG)	Serum potassium (meq/Lt)	CSF analysis		MRI	EMG Types	Final outcome
									Protein mgs%	Cell count			
34	Balasubramaniyan	47/M	879271	Weakness of UL, LL sensory disturbance	-	T_2DM	Orthostatic hypotension	4.9	705	15	-	AMSAN	Spontaneous recovery
35	Rusiya	22/F	881321	Weakness of UL, LL, neck muscles	-	-	Atrial ectopics	4.9	600	<10	-	AIDP	Spontaneous recovery
36	Ashokan	45/M	881505	Weakness of UL, LL, neck, bulbar, respiratory and ocular muscles, ataxia.	URTI	-	-	4.5	327	26	-	MFS	Spontaneous recovery
37	Arulmary	52/F	882773	Weakness of UL, LL sensory disturbance	-	SHT	Ventricular ectopics	3.5	945	30	-	AIDP	Spontaneous and delayed recovery
38	Anand	28/M	888678	Weakness of UL, LL, neck muscles	URTI	-	-	3.6	632	22	-	AMAN	Spontaneous recovery
39	Munuswamy	65/M	899221	Weakness of UL, LL sensory disturbance	-	-	Orthostatic hypotension Urinary retention	3.5	430	25	N	AIDP	Spontaneous recovery
40	Kumaran	25/M	907968	Weakness of UL, LL, neck, bulbar, and respiratory muscles	-	-	Urinary retention	4.8	1120	34	-	AIDP	Spontaneous and delayed recovery
41	Krishnamoorthy	64/M	909916	Sensory Disturbance	-	-	Sinus Tachycardia	4.7	850	25	-	SENSORY GBS	Spontaneous recovery
42	Kavitha	27/F	912253	Weakness of UL, LL, neck, bulbar, and respiratory muscles	URTI	-	-	4.9	980	30	-	AIDP	Spontaneous and delayed recovery
43	Rengaraj	45/M	917321	Weakness of UL, LL, neck, bulbar, and respiratory muscles, sensory disturbance	GE	T_2DM	Ventricular ectopics	4.3	1442	45	N	AIDP	Residual neurological deficit
44	Shanthi	35/F	923580	Weakness of UL, LL	-	-	-	4.2	540	35	-	AMSAN	Spontaneous recovery
45	Karthikeyan	29/M	926360	Weakness of UL, LL, neck muscles	-	-	Orthostatic hypotension	3.6	756	30	-	AIDP	Spontaneous recovery
46	Saroja	38/F	929302	Weakness of UL, LL, neck, bulbar, and respiratory muscles	-	-	-	3.9	-	-	-	AIDP	Expired
47	Murugesan	58/M	931978	Weakness of LL	-	-	-	4.2	630	26	-	AMAN	Spontaneous recovery
48	Neelavathy	28/F	936085	Weakness of UL, LL	-	-	-	5.1	456	35	-	AMAN	Spontaneous recovery
49	Pattu	55/F	941002	Weakness of LL	-	-	-	3.8	864	30	-	AMAN	Spontaneous and delayed recovery
50	Rajendran	37/M	942019	Weakness of UL, LL, neck muscles	-	SHT	-	3.6	1240	25	-	AIDP	Spontaneous and delayed recovery